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The Relation of Wild Animals to Certain Diseases of Man.*

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In the highly civilised state to which the majority of mankind has attained at the present day, it is only in the tropics, where he still exists in a more or less primitive state, that an intimate contact with the indigenous fauna is possible; and it is therefore mainly in tropical countries that we find him a prey to disease germs which normally infest wild animals. Probably, at one time, some of these parasites were more widely spread than at present; but the exact manner in which they originated still remains a matter for speculation. It is probable, too, that primarily their virulence was much greater, but that, in process of time, each species of animal became tolerant of each particular parasite, so that eventually a state was attained in which host and parasite existed on terms of harmony; this is a condition such as we understand by the "reservoir" state; but it is one in which the parasite retains its virulence for non-immune individuals.

There is hardly a subject which so strongly supports the Darwinian hypothesis of the origin of man as this, and it is one which Darwin himself, had he been familiar with the details, would doubtlessly have exploited to the full. That man is in fact but part and parcel of the great scheme of nature and heir to the parasitic infections of animals around him, and a victim to parasites which fulfil their purpose by destroying or checking the numbers of immigrant species, appears to us now to be a self-evident fact.

For the passage of these disease germs from one host to another prolonged and intimate contact is necessary, but when once this chain of contact is broken, the parasite is no longer able to attain the different stages necessary for its evolution and must perish. But in the process of civilisation certain animals such as the ox, the pig and dog, have during domestication inherited diseases from their wild ancestors and have continued to transmit them to man, who in turn has become, in some cases, the necessary intermediary host. The theories as to how these disease germs arose in the first instance, and how they became adapted to man, we will discuss at a later stage.

Trypanosomiasis.

The trypanosomes are blood parasites of many diverse orders of the animal kingdom; they attain their greatest development in the continent of Africa, where they exist as apparently harmless commensals in the blood of the larger ruminants, especially the antelope of that country. These organisms have become adapted to very varied conditions, and species peculiar to birds, reptiles, fishes and amphibia are known.

In Africa, man has become infected with only two species which infest mammalia, *viz.*, *Trypanosoma gambiense* and *Trypanosoma rhodesiense*. In South America a perfectly distinct species, *Trypanosoma cruzi*, has been found parasitic in man in certain limited areas in Brazil and Venezuela.

Trypanosoma gambiense is responsible for the best known form of sleeping sickness in man; it is indigenous and peculiar to Central Africa, where it has spread as an epidemic over a vast area during the last half century. The range of this disease is coterminous with that of the tsetse-fly, *Glossina palpalis*, which acts as its definite host. Under laboratory conditions, this trypanosome can be conveyed to most domestic animals, to monkeys and the larger antelopes, but there is a good deal of evidence that at the present day it is essentially a human infection and is spread by the agency of man to man. There is, however, one antelope which, from its habits and its range, is admirably adapted to act as a reservoir of *Trypanosoma gambiense*. This is Speke's sitatunga (*Limnotragus spekei* [2], or *Tragelaphus selousi*), of which naturally infected individuals have been found by Duke [3] on the Sesse Islands in Lake Victoria. This very handsome antelope stands 36in. at the withers. The buck possesses fine spirally twisted horns. In ground colour it is of a uniform greyish-brown, but the head is adorned with white ocular and cheek spots and a white chin. The type species comes from the Sesse Islands, but minor variations have been traced throughout its range which extends through the lakes and swamps of Eastern and Central Africa. This species, which was discovered originally by Speke in 1861, is a very wary antelope, and as it lives in dense and impenetrable papyrus, it is rarely seen or shot by Europeans. It occurs in the greatest numbers on the islands already mentioned, which from the year 1900 onwards were ravaged by sleeping sickness to such an extent that in 1909 the inhabitants were moved to the mainland. Five years [4] subsequently the tsetses of the island were still found to be infected with *Trypanosoma gambiense*, a fact which was attributed by Duke to the presence of infected antelopes of this species. The hoofs of this antelope are long and widely splayed, an admirable adaptation to its habitat, but since the removal of the population from the islands already referred to it has come to live on dry land and its hoofs have become much shorter and modified accordingly.

The sleeping sickness of man produced by *Trypanosoma rhodesiense* does not differ essentially from that produced by the former species. On the whole the

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illness runs a much more rapid course, and is accentuated by severe febrile paroxysms. Fatal symptoms usually supervene within a year of infection. The organism is much more resistant to drug treatment, especially the arsenical compounds, which have been found efficacious in *Trypanosoma gambiense* infections. This disease has a curious distribution in Central Africa, for it corresponds very closely to that of *Glossina morsitans*—the tsetse of the big game; but it never appears to spread in man in epidemic form. This sleeping sickness occurs in North-Eastern Rhodesia, especially in the Luangwa Valley, in the south-eastern portion of Tanganyika territory, in Nyasaland in the region south and west of the lake. Whilst present in the blood of man, *Trypanosoma rhodesiense* is indistinguishable from *Trypanosoma gambiense*, but it was differentiated by Stephens and Fantham in 1910 by the fact that when inoculated into laboratory animals, especially the rat, a change takes place in the trophonucleus, which assumes a position close to the kinetonucleus or sometimes posterior to it. Subinoculations into laboratory animals are invariably successful and run a rapid and fatal course, in contradistinction to what happens in the case of *Trypanosoma gambiense*.

The part played in the dissemination by the wild game of this parasite to man has been the subject of a great diversity of opinion.

The trypanosome of the big game in this area, *Trypanosoma brucei*, belongs to the polymorphic group of trypanosomes and is indistinguishable, morphologically, from *Trypanosoma rhodesiense*. Yorke, Kinghorn and Bruce believe that under certain conditions *Trypanosoma brucei* can be inoculated into man and produce a *rhodesiense* infection, but others, notably Kleine, Taute and Huber, by experiments upon themselves, either by injecting themselves with *Trypanosoma brucei* infected blood, or through being bitten by infected *Glossina morsitans*, without contracting the disease, consider them specifically distinct. It is, however, clear that a large proportion of the game must be infected with *Trypanosoma rhodesiense*. Waterbuck (Weck [5] 1914) have been infected with human *Trypanosoma rhodesiense* and have shown no signs of disease, though this animal's blood produced in turn a fatal infection in monkeys and dogs.

Three species of antelopes are usually regarded as reservoir hosts of *Trypanosoma rhodesiense*. The handsome common waterbuck (*Cobus ellipsiprymus*, *Ogilby* [6] has a wide distribution in Central Africa; its range extends from the Limpopo River northwards to Tanganyika Territory and Kenya, as far north as Somaliland; on the west it is replaced by allied species. The waterbuck is readily distinguished from others by the white ribbon which passes over the rump and is carried down the thighs on both sides. Although it has the appearance of a clumsy animal, it becomes elegant and sprightly when excited. It can travel at a fast gallop and is difficult for man to follow, though falling an easy prey to lions and leopards.

The waterbuck is always found in greatest numbers on large swampy plains overgrown with coarse grass, tall reeds and papyrus, where in the wet season it is impossible to get at. It revels in almost inaccessible swamps where only elephants, buffaloes and reedbucks care to stay. For this purpose Nature has provided the waterbuck with a tougher hide and coaser hair than any other of its kind, so as to protect it against the sharp fronds of papyrus and other water plants. From the nature of its habitat it will be understood that the extermination of the species would form an almost impossible task.

The reedbuck (*Cervicapra arundinum*, Bodd) [7] is a comparatively large antelope measuring some 36in. at the withers, and in general colour greyish-fawn. It has a thick and bushy tail reaching half-way to the hocks, and handsome evenly divergent horns which curve backwards and upwards. The female resembles the male, but is hornless. There are at least five species of varieties of the reedbuck. Rare in the Transvaal and Bechuanaland, it is still plentiful around Lake Nyasa, and extends as far north as Mozambique and on the west to Angola. This antelope is easily approached, its gallop is slow and irregular, and it is said to be one of the easiest of the South African antelopes to shoot. When alarmed the herd give vent to shrill screams and bound off, kicking up their hind legs and tossing their tails like rabbits. It has not been found possible to keep this species in captivity.

There are many species of *Duikerbuck*; the majority are at home in the dense bush of Western Africa. The common duiker, *Cephalopus grimmi* (Linn) [8] was originally described by Grimm in 1686, and has a wide distribution throughout the *rhodesiense* sleeping sickness area, being found throughout South Africa, along the east coast as far north as Somaliland. It stands 23in. in height, the general colour of the body being pale greenish-brown. Horns are normally present only in the male, in whom they attain a length of 5in. This antelope is called the "duiker," or diver, not from its habit of going into water, but from its manner of ducking and diving quickly into bushes when alarmed. In the Cape it is frequently kept in a semi-domesticated state around farmhouses, but does not thrive in captivity in this country.

South American Trypanosomiasis.

Caused by *Trypanosoma* (*Schizo-trypanum*) *cruzi*, this disease runs a very different course, and has an entirely peculiar natural history. This parasite causes a chronic or acute disease in children, especially in certain districts of Brazil and Venezuela. The symptoms partake for the most part of a myxœdematous [9] character, with febrile disturbances and enlargement of the thyroid gland. The chronic stage may also be seen in adults, when it is characterised by organic syndromes, due to the involvement of the heart muscle, suprarenal glands, or nervous system. The parasite occurs in the blood-stream in forms which are said to represent young individuals which have just escaped into the circulation, and in broader

ones representing an older generation. Multiplication of the parasite by leishmania-like forms takes place in most of the tissues of the body, especially in the skeletal and cardiac muscles.

The infection is disseminated by curious reduviid bugs, the most important of which is *Triatoma megista*; this bug constitutes the definite host of the parasite. A large proportion of these insects are found to be naturally infected; they live entirely on wild animals, such as armadilloes, whose burrows they frequent, but certain species have become domesticated. Both the larvæ and the nymphs of this insect can become infected with *Trypanosoma cruzi*. Considering the wide range of these bugs, and the facts (1) that certain species are found naturally infected with this trypanosome in Argentina, (2) that an allied form, *Triatoma protracta*, is found in a similar condition in the United States, (3) the ease with which the parasite can develop in other insects (*Cimex* and *Ornithodorus*),—it becomes difficult to comprehend the limited geographical distribution of this disease as it occurs in man. The trypanosome is probably under natural conditions conveyed to animals upon which the *Triatoma* feeds, and they therefore constitute the reservoir host. The most important are the armadilloes. The long-tailed armadillo (*Tatusia novemcincta*) [10] appears to be the natural host of *Trypanosoma cruzi*. This species inhabits Mexico and Central America, where it lives in burrows, but in Nicaragua is often kept tame in houses to protect them from invasion by ants. In Guatemala it abounds in primeval forests up to an altitude of 5,000 ft.; there it is much hunted for its flesh, which Dampier described as very sweet and tasting like that of a land turtle. When disturbed this armadillo rolls itself up like a hedgehog. Armadillo burrows are found underneath the houses, and in the immediate vicinity of these, cases of the human trypanosomiasis were originally discovered in Brazil.

The "peludo" (*Dasypus sexcinctus*) [11], a much smaller animal, 18in. in length, common in Brazil, is an omnivorous creature, particularly fond of carrion. It is said to possess a faculty of burrowing up to a decaying carcase, in much the same manner as that of the ground beetle. *Dasypus* has a very short tail, which is shielded by distinct rings near its base.

The other natural hosts of *Trypanosoma cruzi* are entirely different animals. The first of these is *Azara's opossum* (*Didelphys aurita*) [12], a marsupial which has a very extensive range in South America, from Uruguay to Costa Rica. This opossum creates the same havoc amongst poultry in these countries as does the fox in Europe. Sleepy and drowsy in the daytime, the opossums live in the roofs of houses in intimate contact with man, rarely showing themselves in the open. In the towns there are few houses which are not infested with these hideous beasts. Often the stranger is roused from his slumbers by the sound which is caused by these animals running over the roof, upsetting in their course dishes and other household utensils. If he makes inquiry as to the cause of the alarm, he recieves as an answer "Señor es el zoro" (zoro is a fox in Spanish).

The fourth reservoir host of this interesting parasite is a rock cavy (*Cavia* (*Cerodon*) *rupestris*) [13], which is indigenous in Brazil in rocky situations. It is usually found near rivers, but always in the higher parts of their course, where it seeks its retreat in holes and amongst rocky boulders. Superior in size to most other cavies, and standing higher on its legs, it is remarkable for the softness of its fur. It is 14in. in length and its flesh is said to be well flavoured and much sought after by the Indians. Brumpt and Gomes found infected triatomas in the burrows of this rodent in a deserted spot ten miles from any human habitation.

The history of this trypanosome is of comparatively recent date and is remarkable, not only for its peculiar life-history, but from the circumstances surrounding its discovery by Chagas in 1909. This observer first found trypanosome-like flagellates in the hind gut of triatomas, and Cruz, on causing these insects to bite a monkey, subsequently found trypanosomes in its blood. It was this discovery which led Chagas to search for the trypanosome in the blood of man; in this he was successful in discovering it in the blood of individuals living in the very houses from which the infected triatomas were at first derived; there also he found the parasite in the blood of cats.

Leishmaniasis.

Parasites of the genus *Leishmania* are intracellular organisms, which, from a study of their structure inside the human body, as well as of their development into a flagellated stage in artificial media outside the body, are shown to be in close affinity with the trypanosomes. The *Leishmania donovani* is the parasite responsible for a generalised disease in man, known as kala-azar, which has a peculiarly restricted geographical distribution, being found widely spread in India (especially in Assam and the United Provinces) China (Central Provinces and the Valley of the Yangtse Kiang), Central Africa (Sudan, West Abyssinia and Tchad territory), along the shores of the Mediterranean basin, and in the Ægean Islands. In this latter area it is associated with a similar infection in dogs. The peculiarities of its distribution would seem to indicate that it is in some manner connected with the presence of some free-living reservoir host. The parasite can be communicated, though with difficulty, to dogs, cats, jackals, monkeys, rats and mice, but it is only lately that a really susceptible animal has been discovered. Smyly and Young have found that the hamster (*Cricetus griseus*) [14] is very readily infected; that in these animals an enormous proliferation of the parasite takes place. Apparently, in spite of the great numbers of the parasites, the health of these animals is not seriously affected. It therefore appears possible that this small rodent, or some similar animal, constitutes the reservoir host of the *Leishmania*, and that the actual transference of the parasite to the human body takes place through the agency of a small biting fly, such as the sand-fly, *Phlebotomus*.

The hamster, *Cricetus* (*Cricetulus griseus*, Milne-Edwards) [15] is a little species of the size of a field-mouse, and is common in the country districts round

Peking and extends into Chinese Mongolia. This little creature makes extensive burrows, frequenting cornfields and destroying quantities of grain. Greyish-brown in colour, it is pale beneath, has large black eyes, and a median dorsal brown stripe.

Weil's Disease and Infections with Leptospira.

A severe and fatal form of jaundice, associated with fever and a high mortality, has been known in Europe since it was first described by Weil in 1886. Its close resemblance on clinical grounds to yellow fever has long been recognised, so much so that, long before the true ætiological agent was discovered, it was known as Mediterranean yellow fever. The parasite, *Leptospira icterohæmorrhagiæ*, was discovered in 1916 [16] by several Japanese investigators, and they were able to reproduce a typical jaundice in guinea-pigs. It has since been shown that this *Leptospira* is normally a parasite of a rat (*Rattus norvegicus* and *Rattus alexandrinus*) in Europe, and of the field vole (*Microtus montebelloi*) in Japan. In these animals apparently the organism lives in the urinary tubules of the kidney and is excreted in the urine. Thus it happens that large numbers of apparently healthy rats harbour this parasite. Foulerton [17] and, later, Stephenson and Balfour [18] found that no less than 22·6 per cent. of London rats are thus affected. Other wild animals can contract the infection as well. Our honorary secretary, Mr. George Dunkin [19], recently found a young fox naturally infected near the laboratory at Mill Hill. This disease may also possibly be conveyed to carnivora in captivity through eating infected rats. Apparently this recently occurred in the London Zoological Gardens, but since Dr. Vevers has forbidden the feeding of these animals with rats the disease has disappeared. Last year it was proved by Okell [20] and others that a naturally acquired typhoid-like illness known as epizootic jaundice in foxhounds is also due to the same parasite. To add to the interest of this subject it has been found that spirochætes of the *Leptospira* type are widely distributed in water [21], especially that of contaminated wells and rivers, and that small outbreaks of Weil's disease have recently been reported in man from Scotland (East Lothian and Edinburgh) and the Midlands. The probability is that this disease is normally conveyed by infected water contaminated by rats, which constitute the reservoir host of the disease. It is a matter of considerable interest, and a subject for speculation, as to why the closely related *Leptospira icteroides*, the organism of yellow fever, should appear to be a parasite of man only. Arguing by analogy from what we know of Weil's disease, which yellow fever so closely resembles in its clinical course, one would surmise that this organism was widely distributed amongst monkeys and other small mammals in those countries (South America and West Africa) in which yellow fever is endemic. The reservoir host, if other than man himself, has still to be discovered.

There still remains to be mentioned a leptospiral disease which has recently been described. This is the seven-day fever of the Japanese. The organism was first discovered in 1918 [22] by Ido, Ito, and Wani and named *Leptospira hebdomadis*; it is apparently a parasite, like *Leptospira icterohæmorrhagiæ*, of the urinary tract of the common field vole, *Microtus montebelloi*, Milne-Edwards. Very little is known about the fever in man, except that it lasts seven days and does not appear to be a fatal disease.

The *Microtus* [23], wrongly called in text-books a field mouse, is really a stump-tailed vole, which is common in country districts in Japan, and is closely allied to similar voles which occur in China and Tibet. There is little to be said about its habits, except that it burrows and feeds upon roots and grain in much the same manner as do similar creatures in other countries.

Rat-bite Fever.

This is a world-wide disease, but it appears to be specially common in Japan.

Following on the bite of an infected rat, sometimes after a prolonged incubation period, a lymphangitis supervenes which implicates the glands of the area involved. Then follow many short attacks of fever coinciding with an exacerbation of local symptoms.

The organism, *Spirillum minus*, was formerly thought to be a spirochæte, is very minute, 1·5 μ to 6 μ in length, and can be found in the blood, as well as in the lymphatic glands, during the height of the disease. This fever is transmitted by the brown rat (*Rattus rattus and Rattus alexandrinus*), and the organism is probably identical with *Spirillum laverani* and with *Spirillum muris*, which are commonly found in the blood of rats and mice. It is said that the disease can also be conveyed by the bite of a ferret or by that of the domestic cat. The wild cat has also been incriminated. *Felis catus*, Linn. [24], is said to still exist in the Highlands of Scotland in a purely wild state, but this statement is doubted by many authorities, as it appears to have intermingled with the house cat wherever that familiar animal has penetrated.

Rocky Mountain Spotted Fever.

This is not a disease of the tropics at all; it is, however, peculiarly associated with the presence of wild animals. A typhus-like disease with a high mortality rate has long been known as "spotted fever," "blue disease," and "black fever"; and to be endemic in limited districts of Montana and Idaho and Wyoming. It has also been reported from the States of Utah, Nevada, Oregon, Colorado, and Washington. This fever is virulent, especially in the spring months of the year in the district of Montana known as the Bitter Root Valley. The causative organism belongs to that rather nebulous group, the *Rickettsia*, and is known as *Dermacentoxenus rickettsi* [25]. It is conveyed by the bite of a tick peculiar to that region, known as *Dermacentor venustus*. It is only the adult tick that attacks man, but it commonly infests the Rocky Mountain goat, the badger, lynx, bear, coyote, and other animals that inhabit that region. The larval and nymphal stages of the tick develop principally on the ground squirrel (*Citellus columbianus*) and

the woodchuck (*Marmota flaviventer nosophora*, Howell). The last two rodents are considered to be the main reservoir hosts of the *Rickettsia* and, although a certain proportion are capable of infection in the laboratory, it is thought that they may develop the disease in a mild form under natural conditions. Of the larger animals the tick most favours the Rocky Mountain goat (*Oreamnus montanus*) [26]. It is not really a goat at all, but is most nearly allied to the serows, a rare animal at the present day, as it only exists where strictly preserved. The American badger (*Taxidea taxus*) resembles its European relative, with the difference that its diet is more carnivorous. The black bear (*Ursus americanus*), and the lynx (*Lynx uinta*) hardly call for comment. The coyote (*Canis latrans*, Say) is a small slinking wolf-like creature which differs from its European congeners in living in burrows in the plains.

The Columbian ground squirrel (*Citellus columbianus* Ord) [27], is closely allied to the chipmunk, and is an intelligent little creature like its near relative ; it lives amongst the birchwoods, where it digs its burrows in such a manner as to avoid attracting the attention of its enemies. In order to do so it removes the earth in its cheek pouches and carries it to a considerable distance. In winter the ground squirrel hibernates and in this state it probably harbours the virus of Rocky Mountain fever, so that it is able to convey the disease shortly after its spring awakening.

The woodchuck (*Marmota flaviventer*) is really a marmot, about 2ft. in length ; it is a thick-set creature with short legs and a stout bushy tail. It is grizzly or yellowish-grey in colour and hibernates all the winter long. Being vegetarian in diet, it lives in woodlands, never in the open plains, and is said to be the least industrious of all animals, to show energy only when digging its burrows, which may be 50ft. or more in length.

Plague.

Plague is essentially in the first place a disease of rodents and not of man. As Macarthur has so lucidly pointed out recently, much suffering would have been saved in the City of London had this been recognised in the Middle Ages.

Although the rat is the main reservoir of plague, many other rodents in many other parts of the world play a not inconsiderable part.

There are, as is well known [28], two species of domestic rat, of which the black rat (*Rattus rattus* Linn.) is the more important in the spread of the disease. The old English rat, as it is called, is rapidly vanishing from this country, being ousted by the more virile North European species (*Rattus norvegicus*, Berkenhout), which was apparently imported into Western Europe in the year 1716 as a result of a visit by the Russian Fleet. The black rat in India, in Burma, and in fact in the tropics generally, lives more closely in association with man ; it is essentially an arboreal or climbing animal and rarely burrows ; hence, when it infestes buildings or huts, it is found in usually the walls, ceilings or roof. It drinks little and seldom enters the water voluntarily and it is the common rat on ships. In most cases it reaches or leaves the ships by clambering along the cables whilst the ships are in dock. Its dietary consists mostly of grain, and it is a much cleaner feeder than *Rattus norvegicus*. In appearance it differs also from *norvigecus* in its more slender build and large transparent ears. The weight of the adult is 8 oz.

There are several races of *Rattus rattus* which have been distinguished. One variety with reddish-black and pure white underparts is known as *Rattus rattus frugivorus* and is common in the Mediterranean area. The Alexandrine rat (*Rattus rattus alexandrinus*), with brownish-grey back and dingy coloured abdomen, is found in Asia Minor and North Africa. *Rattus rattus rufescens* is the common Indian house rat and is distinguished by its smaller size, its long tail and the spinous hairs with rusty tips on its abdomen. These rats breed throughout the year, gestation lasting twenty-one days, the average litter being five or six.

The brown rat (*Rattus norvegicus*) is distinguished by its larger size, its heavier and clumsier build and by its blunter muzzle. The ears are small and opaque the tail noticeably shorter than the head and the body. The weight of the adult is about 17 oz. This rat is now developing a melanistic or black variety which was originally described in Ireland. Its home was originally temperate Asia. It is a water-loving and burrowing animal and is consequently far less agile than the former species, but it is far more voracious and cunning and is possessed of greater strength and fecundity. Usually exhibiting a shyness of man, it is under normal conditions a less important carrier of plague than is *Rattus rattus*. *Rattus norvegicus* is a very rapid breeder, the litter varying from six to twenty-three, the average from eight to ten.

In various parts of the world other rodents are capable of replacing the rat in the spread of plague. In California, McCoy has shown that the Californian ground squirrel plays an important part, and that the plague bacillus is transferred by the flea (*Ceratophyllus acutus*) to rats and so to man. This ground squirrel (*Citellus grammurus beecheyi*, Richardson) [29], is in reality a spermophile and lives in burrows. In Southern Russia and the Caucasus other ground-squirrels (*Citellus*) are said to carry plague. These rodents are really a connecting link between the squirrels and the marmots. They are generally to be found in prairie-like regions, where they form an intricate system of burrows, at the mouth of which they are commonly to be seen standing upright and motionless. The common European species is *Citellus citellus* Linn., and is known in Russia as the " suslik." In Mongolia a subspecies known as *Citellus dauricus* plays a similar part. These animals hibernate throughout the winter and are responsible for the summer outbreaks of pneumonic plague in Mongolia and Manchuria. In these regions, too, the common house mouse, *Mus musculus*, is held to be responsible for winter outbreaks of plague, though it does not appear to play a dominant part in other regions. In North China, Siberia and Manchuria the Mongolian marmot or " tarabagan " (sometimes called tarbagan,

or sarabagan (*Marmota bobak*, Müller) [30] has been held to be responsible for many serious outbreaks of pneumonic plague. These marmots are of a large size and strong build. They are characterised by the rudimentary character of the thumb, their small eyes and ears; the tail is bushy and comparatively short. In length (exclusive of tail) they are 15 to 18 in., and 9 to 12 lb. in weight. They make large, deep and intricate burrows where they hibernate. The mounds of earth thrown up by generations of tarabagans are known as "bootans," and are characteristic of the region they inhabit [31]. The flesh of the tarabagan is eaten by the Mongols, or "Buriats," and its fur is in much demand, being sold after drying and curing as imitation sable or seal [32]. This creature is credited with the faculty of harbouring the plague bacillus in its body during hibernation, and thereby causing outbreaks of pneumonic plague amongst the hunters, who live under miserable conditions huddled together in underground hovels during the autumn and winter. The "Buriats" are said to be well aware of the danger of handling sick tarabagans, the infected animals being recognised by ceasing to bark and by being ejected by their healthy companions when they attempt to crawl back into their burrows.

In India, besides the rat, an insectivore, the musk shrew, *Pachyura cærulea* [33], plays a part in conveying plague. This shrew is about 6in. in length, with a tail of 3½ to 4in. It is coloured uniformly with a bluish-ash or pale grey hue, slightly tinged with red, the naked parts being flesh coloured. Commonly called the "musk rat," it frequents houses at night time, running about the rooms hunting for cockroaches or other insects, and uttering, while so doing, a sharp, shrill cry. It is popularly believed in India that the musky odour emitted by this shrew possesses such volatile and penetrating powers that it will actually permeate the cork of a bottle and taint the fluid within.

The bandicoot rat (*Bandicota gigantea* [34]) is also susceptible to plague. This is a large rat 15in. in length, with a tail of 13in. and weighing about 3 lb. when fully adult. The incisor teeth are peculiar, being olive green at their base and yellow at their extremities. It is a well-known rat throughout India and Ceylon; being of a large size it shows fight when cornered and grunts like a pig. Where European gardens are cultivated, as in Newera, Eliya, Ceylon, it causes considerable damage amongst the potatoes and peas.

In South Africa, plague [35] [36] [37] has spread amongst a number of rodents. When first introduced in 1901 it was apparently confined to the domestic rats. Further outbreaks in 1903, 1914 and 1918, led to the suspicion that other rodents were concerned. Some of them are responsible, as is the Cape hare, for transmitting the infection over a considerable area, but the chief part is played by small rat-like animals known as the gerbilles; of these there are numerous species in South Africa.

The first is known as Lobengula's gerbille, *Taterona lobengulæ* [38]. Gerbilles are distributed all over South Africa south of the Sahara. They are all very closely related and are probably equally susceptible to plague. This particular species has been singled out by the health authorities in South Africa. It receives its specific name because it inhabits the former territory of Lobengula, the Matabele king. It is about 5½in. from the nose to the root of the tail, the latter being 1in. longer than the body. The fur is light fawn on the back, finely grizzled with dull black; the sides are pale fawn, the under parts are white and strongly demarcated. Although a gerbille, this particular species has the habit and structure of a rat. Other species probably involved are known as the Cape gerbille (*Taterona afra*) and Brant's gerbille (*Taterona brantsi*).

The white-nosed rat (*Rattus coucha*), sometimes called the multimammate mouse, is in appearance something between a rat and a mouse, and living in close association with the gerbilles, it is also frequently attacked by plague. This species is distributed throughout South Africa, and its range extends beyond the Equator to the Sahara. It is found in situations which afford abundance of cover, such as thick scrub. It is a house dweller and is often trapped in buildings and outhouses. The tip of its nose, as its name indicates, is white.

The Springhaas, or Cape jumping hare (*Pedetes caffer*), is a much larger animal, the size of a rabbit, and by far the largest of the jerboa family. It is common throughout the Cape Province. It is sociable in its habits and forms an intricate series of burrows in which a good many families take up their abode. When alarmed and travelling at full speed, it races like a kangaroo, each leap covering 8 or 9 ft. It is nocturnal in its habits and multiplies very rapidly. It has many natural enemies and is preyed upon by the Cape eagle owl. This species can travel twenty to forty miles in a single night; on this account it is of importance in the spread of plague.

Carnivores which prey on these rodents, especially when sick, such as the yellow mongoose (*Cynictus penicillata*) and the suricat (*Suricator suricator*), are also found to die of plague; when the fæces contain the remains of gerbilles, which they feed upon only when they are sick, it is taken by the health authorities as an indication that plague is endemic in that particular area.

In Senegal and West Africa a shrew (*Crocidura stampflii* [39]) is said to play a part in the dissemination of plague. It lives in native houses and probably has much the same habits as those of the Indian musk rat and plays a similar rôle.

In England [40] the rabbit and hare were found naturally infected with plague in Suffolk in 1910.

Tularæmia.

Tularæmia is a plague-like disease which has been known since 1912 to affect jack-rabbits and other rodents, especially ground-squirrels, in California. Unfortunately it is communicable to man, in whom it causes an intractable illness.

Tularæmia [41] in man is a disease of the rural population, particularly in field workers, and is recorded amongst dealers who handle infected jack-rabbits. The organism, *Bacterium tularense*, is not easy to cultivate on artificial media: it is extraordinarily infective and many laboratory workers have contracted the disease. The infection is transmitted, from one infected jack-rabbit to another, by a blood-sucking fly, *Chrysops discalis*.

The jack-rabbit (*Lepus campestris*, Bachman) is a very familiar species in America. It is a large hare-like creature with long hind-legs and ears. In the northern parts of its range its coat turns white in winter, but further south this change is partial, or does not occur. Like our hare, it lives in the open and depends for safety on its protective coloration, speed and acute hearing. For its home the jack-rabbit has an open form beneath a bush or clump of weeds. Large numbers of these animals are killed annually in drives, as many as 20,000 having been accounted for in one day.

The ground-squirrel (*Citellus grammurus beecheyi*), which also forms a reservoir of infection, is the same species as that concerned in the spread of plague in California.

The gophers (*Geomyidæ*) form another source of infection. These are curious little animals, characterised by large cheek pouches opening outside the mouth, and they have feet with immense claws for digging. Their bodies are heavy and their movements slow and clumsy. Their habits are nocturnal and they live in communities in burrows. *Thomomys bottæ bottæ* (Eydoux and Gervais), which extends along the coastal region of California, is the species concerned.

Melioidosis.

There still remains one more recently described glanders-like disease in man known as *Melioidosis* [42], which occurs in Burmah and in the Malay States. The organism known as *Bacillus whitmori* occurs as a natural infection in the Malay rat (*Mus griseiventer*, Bonhote), which, in spite of its name, is the local representative of the common black *Rattus rattus*.

I have now summarised somewhat incompletely, I fear, the state of our knowledge regarding the wild animal reservoir hosts of human disease in the wilder portions of the earth. There still remain the helminthic infections that I do not propose to deal with in detail, but of which I append a list at the end of this paper.

There are other widespread and almost universal infections, such as malaria, in which the parasite, though indigenous in most parts of the tropics, is peculiar to man, and to man alone. Strange to relate, human malaria parasites have never been found in any other vertebrate host, not even in the larger apes which harbour, it is true, their own particular species of *Plasmodium*. From this circumstance I think we may fairly infer, from what we know of the natural history of other protozoal infections, that primæval man must have acquired his *parasite* from some reservoir host.

If it is usually considered permissible for ethnologists, dealing solely with his more durable remains, to speculate upon the heredity of man, so it is equally permissible for the parasitologist to speculate upon the probable evolution of man's diseases. It may be that the original reservoir host of malaria was some hoary and hairy simian anthropoid, one of the missing links long since extinct and whose bones lie buried deep in the alluvial earth of Central Africa. But though the anthropoid became extinct, his parasite did not and was transmitted as a sort of heirloom to his more intelligent and highly developed descendant—man.

But whence did these remarkable parasites arise, organisms which have such an intricate life-history? Did the malaria parasite first exist in the mosquito and later in man, or vice versa? Were the trypanosome and tsetse on intimate terms long before man was swinging in the tree tops of the great African forest? I fear that we have no very exact *data* upon which to base any reasonable hypotheses. One can readily understand how a free-living organism can readily adapt itself to the life of another animal (there are plenty of instances of this in Nature), but it is more difficult to explain how it can accustom itself to hosts so entirely dissimilar in structure and habits, as man and the tsetse, or man and the mosquito, and as to how the parasite came to lead the entirely different life-cycles within the vertebrate and invertebrate host.

Possibly the blood protozoa of man and animals were at one period parasitic in the latex of certain plants, and, through being constantly ingested by insects which feed on these juices, they eventually came to adapt themselves to a semi-parasitic existence within the alimentary canal of the insects. In course of time, and in process of evolution, the predatory instincts of a more adventurous bug led it to feed upon the warm blood juices of rodents and other mammals. Ingrained habits may change; thus the Kea parrot of New Zealand (*Nestor notabilis*) found the liver of sheep imported into that country more succulent than the acrid fungi upon which it was wont to feed. It may be so, by constant close association of insect with food supply, that the primitive parasite followed suit and came to pass part of its existence first in the one and then in the other.

Certain discoveries which have been made in recent years support this hypothesis.

Flagellates of the *Leptomonas* type, closely allied to leishmania, have been found by Lafont, Migone (1916), França (1921), and others, in the latex of plants of the genus *Euphorbia*; the second named observer has shown that a plant bug, *Stephanocephalus agilis*, is responsible for the spread of infection from plant to plant in Portugal. The bug, which is nocturnal in its habits, punctures, while feeding, the leaf of the plant in many places and transmits the parasites through its salivary glands. Strong (1924) makes a stronger claim in stating that lizards which devour certain of these plant bugs acquire an intestinal infection with

the flagellate, which then, he asserts, assumes pathogenic properties for monkeys.

Other flagellates which they closely resemble in structure inhabit the intestinal tract of house flies (*Herpetomonas muscæ domesticæ*), bluebottles and even biting flies.

From a morphological standpoint these free-living forms are indistinguishable from the evolutionary stages of trypanosomes and leishmania, the familiar blood protozoa of man.

It has been further claimed by some investigators, notably Laveran, Franchini, and Fantham, that vertebrates, particularly mice, may be easily infected with insect flagellates by inoculation or feeding, and that in some instances a diseased condition resembling kala-azar may result. But this work has not received the necessary confirmation, so that it is only by intensive research and numerous transmission experiments with these lowly forms of life that further light can be shed, not only on the origin of the parasites themselves, but also upon the chequered history of *Homo sapiens* himself.

I must acknowledge, as is only due, my gratitude for the generous assistance I have received in preparing this paper from Mr. M. A. C. Hinton, of the British Museum, Mr. Martin Duncan, of the Zoological Society, and Dr. Daukes, of the Wellcome Bureau.

List of Helminthic Parasites found in Man and Wild Animals.

Trematoda :—

Fasciola hepatica. Wild ruminants, deer and antelopes.
Heterophyes heterophyes. Canis niloctus. Egypt.
Paragonimus westermanii. Tiger and leopard. India.
Schistosoma hæmatobium. Sooty mangabey monkey (*Cercocebus fuliginosus*).
Schistosoma japonicum. Rats (*Rattus*). China.
Gastrodiscoides hominis. Mouse-deer (*Tragulus napu*). Malay States.

Cestoda :—

Cysticercus cellulosæ, larval stage of *Tænia solium.* Wild boar (*Sus scrofa*).
Cysticercus bovis, larval stage of *Tænia saginata.* Giraffe (*Giraffa camelopardalis*).
Hymenolepis diminuta. Rats (*Rattus rattus, Rattus decumanus* and *Rattus alexandrinus*). Mice (*Mus musculus* and *Mus silvaticus*).
Dipylidium caninum. Jackal (*Canis aureus*).
Echinococcus granulosus, larval stage of *Tænia echinococcus.* Wolf (*Canis lupus*). Jackal (*Canis aureus*).
Sparganum mansoni, the plerocercoid stage of *Dibothriocephalus mansoni.* Frog (*Rana nigromaculata*). Snakes (*Elapheclimacophora*). Japan.
Diplogonoporus grandis. Various species of seal. Japan.

Nematoda :—

Belascaris canis. Fox (*Canis vulpes*).
Toxascaris canis. Wild cat (*Felis catus*).
Ternidens deminutus. Monkeys (*Macacus sinicus* and *Macacus cynomolgus*). Nyasaland and Transvaal.
Physaloptera mordens. (*Macacus sinicus*). Nyasaland.
Ancylostoma duodenale. Tiger. India.
Ancylostoma ceylanicum. Civet cat (*Viverricula malaccensis*). Ceylon. Clouded leopard (*Felis nebulosa*). Malay States.
Necator americanus. Gorilla (*Gorilla savagei*). Patas monkey (*Cercopithecus ruber*).
Æsophagostomum apiostomum. Gorilla. Orang-outang (*Simia satyrus*).
Trichuris trichiura. Various old-world monkeys.
Hepaticola hepatica. Rat (*Rattus norvegicus*).
Trichinella spiralis. Wild boar (*Sus scrofa*).
Loa loa. Baboon (*Papio cynocephalus*). West Africa.
Acanthocheilonema perstans. Chimpanzee (*Anthropopithecus troglodytes*). West Africa.
Dracunculus Medinensis. Jackal and leopard.
Gnathostoma spinigerum. Tiger. India. Puma (*Felis concolor*). South America.

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Discussion.

Dr. H. Harold Scott said he was a little surprised that such an important disease as rabies had not been mentioned, but naturally in a paper which had to be of limited length owing to time available, some omissions must be expected. He spoke of two

interesting conditions which might, perhaps, be regarded more as common to man and animals than actually communicated from the latter to the former, though communication was quite possible. The first was a small epidemic he had investigated amongst the hamadryas baboons at the Zoological Gardens, in the course of which sixteen of them had died with intestinal ulceration. From twelve of these he isolated from the bile and from septicæmic abscesses in the lungs and the liver the *Bacillus aertrycke* which was the cause of severe and often fatal lesions in man. The second was the isolation of *Bacillus morgan*, No. 1, from two *Patas cercopithèques* recently dying from enteritis at the Gardens. This organism, as was well known, had been incriminated as one of the causes of summer diarrhœa of children—a disease commonly fatal.

(*Communication to the Discussion made by letter dated January* 19, 1926.)

Mr. EUSTACE MONTGOMERY (Nairobi, Kenya): I wish I could be present for Manson-Bahr's paper, but it is a combination, *i.e.*, wild animals and man, of which I have no personal experience except our pseudo-rabies and trypanosomiasis. The former appears indeed not to be a "disease" of man and the latter could be discussed with advantage, but without finality, for days. What, however, might with advantage be brought out at the discussion is the great anomaly of "game" control in most of our tropical possessions. I am a keen "shikari" and advocate protection against extermination for many reasons, but as practised by a game "department," numbers and range of species are encouraged to expand largely at the expense of man or his domestic stock. In such fortunate countries where there exist no diseases (contagious) of stock, and where grazing, water, etc., suffice for all, no harm is done. But in such as Eastern Africa, the veterinary departments, armed with very elaborate and often most penalising "Diseases of Animals Ordinances," demand of a stock owner that he shall fence his land, brand his cattle, etc., inoculate them against various diseases and so on. In other words, great restrictions are put on domestic mammalia in the interests and needs of disease control. In most countries, for example, no cattle can be taken from one village to another without a written permit specially obtained. Yet we encourage wild mammalia, lacking even the most primitive control of the most uncivilised natives' cattle, to roam at will without fences, permits, brands or inoculations. Wild and domestic are susceptible to the same great East African menace—rinderpest—and are naturally carriers and disseminators; and, without question, inquiry would disclose more than rabies, anthrax, swine fever, horse-sickness as between wild or domestic.

Both are susceptible mammals; ofttimes the wild is the greater menace, and yet for the sake of "protection," as well as revenue from licences to shoot, this extraordinary difference is made between the

(Continued at foot of next column.)

Foot-and-Mouth Disease—Control by Chemotherapy.*

By Colonel G. K. WALKER, C.I.E., O.B.E., F.R.C.V.S., Principal and Professor of Veterinary Medicine,
and
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INTRODUCTION.

The repeated occurrence of outbreaks of foot-and-mouth disease in England of late years has stimulated both public and scientific interest and created a demand for a more economical method of control than that of slaughtering affected and contact animals.

Laboratory research has revealed some valuable knowledge in connection with the transmission of the virus by artificial methods, though no accurate information is yet forthcoming in regard to the way by which natural infection usually takes place. A satisfactory virus having been obtained, the experiments now recorded have been conducted with a definite objective based on the knowledge that the first symptom of foot-and-mouth disease is thermal and precedes the appearance of the primary eruptions. This rise in temperature is very marked when thermometrical readings are conducted, but feverish symptoms are otherwise easily overlooked.

The thermal stage is usually followed by the primary eruptive stage in 8-24 hours.

It was anticipated that if the normal course of the disease could be arrested at the thermal stage by means of a suitable therapeutic agent a practical and inexpensive method of controlling outbreaks might be evolved. In addition, if it were found that the abortive febrile stage, in itself, proved sufficient to produce even a short period of immunity, the method would possess further advantages.

Difficulties have hitherto been experienced in obtaining a sufficiently virulent strain of virus in India for the experiments and disappointments have occurred owing to failure to communicate the disease by means of natural infection and by inoculation. The present knowledge of the manner in which natural

(*Continued from previous column*).

classes. Either the precautions now applied to the domestic are unnecessary or the freedom to the wild to roam and propagate is undesirable. In any case, it is illogical so to make fish and fowl of susceptible species of a common stock.

The remedy is not game destruction, as some would urge, but scientific game protection, as there is scientific stock protection, and this would also encourage enquiry into habits as well as diseases intercommunicable with man and domestic stock. We have many men ideally competent for such observation, and, if they do not want to be bothered to sell licences and to prosecute poachers, there are such departments as Revenue, Administration and Police quite well constituted for this everyday purpose.

* *Veterinary Bulletin, No. 17*, Department of Agriculture, Punjab.

infection is conveyed is so nebulous and cases of foot-and-mouth disease in India are so rarely brought to notice until well advanced that it has so far proved impossible to obtain a satisfactory virus from this source. The variation in susceptibility of Indian types of cattle is another difficulty that has to be faced. This factor was discussed in the report of the Departmental Committee on Foot-and-Mouth Disease appointed in 1912, wherein it was stated that plains animals were unsatisfactory for experimental purposes. Through the courtesy of Sir Stewart Stockman, virus in the form of some pads of infected guinea pigs was recently brought to India with the necessary precautions and was found to be infective for guinea pigs on arrival. The disease was easily passed from one guinea pig to another by means of emulsified pads of infected animals. The same emulsified pads, even when blood of infected guinea pigs was added, proved to be non-infective, however, for Indian plains calves, though the same calves were shown later to be susceptible to an Indian strain of virus.

An opportunity occurred recently to obtain fresh virus from a severe outbreak at Hissar, which was reported by Mr. Branford, the Superintendent of the Government Cattle Farm, to be exceptional in virulence and to be causing mortality. His ready help in the matter is hereby gratefully acknowledged.

The virus used in the experiments was obtained from a cow in Hissar City showing a temperature of 105·4° on the morning of February 1st, by bleeding from the jugular vein, 400 c.c. being taken. At 1 p.m. this cow developed large vesicles on the tongue and on the lower lip. The contents of these were taken and mixed with normal saline solution. The epithelium of the vesicles was removed subsequently and preserved in 50 per cent. glycerine with distilled water. At the time of injection the blood, the diluted vesicle contents and the epithelial material, emulsified in normal saline, were mixed together. A guinea pig was inoculated with this mixture intracutaneously in the pad and developed typical lesions of foot-and-mouth disease. The cow went through a normal course of the disease and recovered.

S. Raizul Hussan, the Assistant to the Professor of Pathology at the Punjab Veterinary College, was detailed to obtain the virus, and his success in this matter and his great assistance in the subsequent experimental work is greatly to be commended and acknowledged.

Experimental Procedure.

In each experiment, with one exception, two animals (weaned calves) were used, one for treatment and one for control. In one case three animals were taken, one being kept as control and the other two treated. In the first experiments hill animals were used on the assumption that they were likely to be more susceptible than plains animals, but later it was found that plains animals proved equally susceptible to this particular virus. After the period of incubation and during the thermal stage each animal to be treated received Lugol's solution of iodine intravenously. With a view to determine the requisite dose various quantities of the solution were tried, starting with what was considered to be the largest dose likely to be tolerated.

During the course of the experiments guinea pigs were inoculated with the virus used and in every case typical lesions were produced.

Dosage and Method of Administration.

The solution used in all the experiments was iodine one gramme, potassium iodide two grammes, distilled water 300 c.c. It was found that doses of over 100 c.c. were not always tolerated, though one animal received 300 c.c. and survived. The dose that is recommended is from 50 to 100 c.c. according to the size of the animal. In one case (No. 155) 30 c.c. proved to be effective. The most convenient method of administration is by means of a syringe. When dealing with restive animals a funnel was found to be inconvenient.

(Details of the experiments made are given.—Ed.)

In order to determine if the rise in temperature conferred immunity, all the treated animals were exposed to natural infection and, in addition, inoculated intravenously with virulent blood at varying intervals ranging from one to twenty-four days after the injection of iodine as noted in the records. In no case did any symptoms of foot-and-mouth disease appear, showing that considerable immunity had been conferred. The infectivity of the virus was verified by obtaining positive results in controls.

Consequent on these results, it was considered advisable to ascertain the effect of injecting Lugol's solution previous to inoculation with virus in order to discover if any protection resulted or if the normal course of the disease was interfered with.

Out of six plains calves four (Nos. 157, 158, 159 and 160) were injected intravenously with 50 c.c. Lugol's solution on February 25th. On the following day No. 157 and two controls (Nos. 161 and 162) were each inoculated intravenously with 20 c.c. citrated virulent blood.

Three (Nos. 158, 159 and 160) were inoculated similarly on February 27th, February 28th and March 1st respectively. The controls (Nos. 161 and 162) developed typical attacks of foot-and-mouth disease and recovered (161 lost twenty lbs. in weight and 162 five lbs.). The treated animals were inoculated respectively with virulent blood, No. 157 one day, No. 158 two days, No. 159 three days and No. 160 four days after the injection with Lugol's solution. Symptoms of foot-and-mouth disease appeared in No. 157 twelve days, in No. 158 eleven days, in No. 159 eight days and in No. 160 four days after inoculation, and in each case the disease ran a normal course.

Discussion.

From the above experiments, it appears that the conclusion is warranted that cattle infected with foot-and-mouth disease can be cured in the febrile stage by the intravenous injection of Lugol's solution of iodine, and that such animals will not develop eruptive lesions if the treatment is adopted in time. Contrary to the findings of the Departmental Committee, it was

observed that there was always a decided rise of temperature before lesions appeared. It appears, moreover, that considerable immunity is attained, but whether this immunity is as active as the immunity which follows an attack allowed to pursue its normal course must be a matter for further experiments. It is well-known that immunity in natural cases is very variable and not often absolute. The period of infectivity, if it exists at all, in animals treated by this method must be very short, as it seems fair to presume that if no eruptive lesions occur the disease is unlikely to be conveyed by natural infection. This point affords a field for some interesting experimental work later.

With regard to the experiments in which Lugol's solution was injected previous to inoculation with virus, it was observed that, although the temperatures of the animals rose to heights varying from 103·6° to 105° and remained in that vicinity for several days, the eruptive stage was much delayed, more particularly in two cases (Nos. 157 and 158).

It appears, therefore, that the intravenous injection of iodine attenuates the virus if the inoculation of virulent blood takes place within two days of the injection. As this observation is dependent on one experiment only it may be regarded as tentative pending the result of further experiments on the same lines.

Recommendations.

Consequent on a consideration of the results recorded, which appear to be extremely significant, and, if confirmed in the field, of far-reaching economic importance, it is suggested that outbreaks of foot-and-mouth disease in cattle may be dealt with in future by treating animals in the febrile stage by the intravenous injection of Lugol's solution of iodine. In a case in which iodine was administered late and vesicles had appeared, treatment was found to a have good effect. On the occurrence of a case of the disease, the ideal method would appear to be to ascertain and record the temperatures of all the contact animals twice daily and treat those which show a rise above normal. If, for certain reasons, it is not possible to take thermal readings, it is suggested that all the contact animals be injected with iodine, but, as the experiments show, no appreciable immunity would be conferred and this method should only be used as a safeguard pending the adoption of the former. Another proceeding worthy of consideration is to inoculate all contact animals with virus and inject them as soon as their temperatures rise with a view to confer immunity.

Cases showing primary vesicles may be regarded as infective; and it has been shown, in one case, that, even so, the course of the disease can be arrested, and therefore, if all contact animals showing temperatures are treated, those which have developed eruptive lesions may be regarded as having completed all the harm they can and need not be slaughtered.

The experiments so far conducted show that animals which have had a rise of temperature and have been treated with iodine become immune for at least twenty-four days. This is very satisfactory so far as it goes, but nevertheless, the advisability of reinoculating the treated animals with further doses of virus with the view of increasing their powers of resistance and extending the period of immunity is a matter for consideration.

Measures directed to the thorough disinfection of all possible or probable sources of infection and the destruction of all soiled fodder, litter and vermin are indicated in order to prevent future infection.

Conclusions.

The experiments now recorded show that outbreaks of foot-and-mouth disease in cattle may be dealt with effectively and economically in future by therapeutic treatment.

It is considered that the importance of the subject justifies the early publication of the results so far obtained in order that further experimental work by others may confirm or dispute the conclusions already arrived at. If the above simple method of treatment, which has so far never failed, is found to be capable of superseding wholesale slaughter, a serious economic problem will be solved. Additional advantages are that the treated animals show no obvious lesions of foot-and-mouth disease, their general health is little affected and there is no appreciable loss in weight and condition.

Iodine is very rapidly eliminated from the system, and the amount used is unlikely to have any harmful effect on flesh or milk. The treatment can be conducted easily by professional men and at relatively small expense.

Organisation of Education of Veterinary Surgeons in Japan.*

The following regulations govern the veterinary surgeons.

Veterinary Surgeons' License Regulations.

(*Law No.* 76.)

Article I. Only those who have received licenses from the Minister of Agriculture and Commerce are permitted to practice veterinary surgery.

Article II. Those who are qualified to receive veterinary surgeons' licenses are as follows:—

1. Those who have passed veterinary surgeons' license examinations and possess the certificate thereof.
2. Those who possess certificates of graduation in the courses of veterinary surgery in a governmental or prefectural veterinary or agricultural school.
3. Those who possess certificates of graduation in veterinary surgery in a public or private school established in compliance with the school regulations which have been approved by the Minister of Agriculture and Commerce.

*Reprinted from "Medical History and Medical Education in Japan," published by Far Eastern Association of Tropical Medicine, Tokyo, 1925. *Vta Jl. Jap. Soc. Vet. Med. Sc.*

4. Those who possess certificates of graduation in schools of veterinary surgery in foreign countries, which are equivalent or higher in the standard in comparison with those of the governmental or prefectural schools of veterinary surgeons or agriculture.

According to the foregoing regulations, the veterinary surgeons are placed under the jurisdiction of the Minister of Agriculture and Commerce and not under the jurisdiction of the Minister of Home Affairs as physicians, dentists or pharmacists. It is not necessary to be a graduate of school of veterinary surgeons in order to receive a license. Those who have passed a certain veterinary surgeons' examination are granted the license. The majority of the veterinary surgeons, however, are graduates of schools of veterinary surgeons.

There are twenty schools of veterinary surgeons, two of which are university standard, three higher school standard, and sixteen the secondary school standard. The last class of the schools was established during the Restoration period, when there was no veterinary surgeon in the country. At that time animal husbandry was just being introduced and animal diseases were also brought in, causing extensive damage. Consequently the government encouraged the prefectures to establish schools of veterinary surgeons as a temporary measure. These schools were established in accordance with this measure. At present the veterinary surgeons are well distributed throughout the country, and it is being recognised that the low grade veterinary schools are no longer needed, and these schools are doomed to closure sooner or later. Many of the graduates of these schools, however, enter higher schools for further study.

There are the following regulations concerning the veterinary schools.

Regulations on the Veterinary Schools.

1. The number of instructors and their qualifications: There shall be more than three instructors in a veterinary school, one of which shall be a graduate of a university or have equivalent attainments.

2. The following courses must be given: Botany, zoology, physics and chemistry, obstetrics, general pathology, animal plagues and veterinary police, anatomy and its practice, pathological anatomy and its practice, physiology, breeding and hygiene of animals, pharmacology, horse shoeing and its practice, internal medicine, clinical practice, surgery.

3. The courses must be extended over three years.

There are the following regulations for the veterinary license examination.

The examinations are given in the following courses:—

Anatomy, physiology, pharmacology, internal medicine (including epizootiology), surgery (including ophthalmology and obstetrics), veterinary police (including veterinary hygiene), zootechny, clinical diagnosis and surgical operation, horse shoeing and its practice.

	NAME OF SCHOOL.	Location.	Date of founding.	Term of School year.	No. of Instructors.	No. of Graduates.	Entrance Qualification
Governmental.	Dep. of Veterinary Science, Faculty of Agric., Tokio Imp. Univ.	Tokyo ...	1877	3	27	262	Graduate of higher schls.
	The Second Division of Depart. of Zootechny, Faculty of Agric. Hokkaido Imp. Univ. ...	Sapporo	1907	3	35		Gradute of preparatory course, higher schools.
	Subsidiary Course in Veterinary Medicine, Faculty of Agric., Tokio Imp. Univ.	Tokyo ...	1899	3	12		Graduate of secondary school.
	Veterinary Division Agric. and Forestal College of Morioka...	Morioka	1901	3	21	351	do.
Public.	*Veterinary Division.* Miyagi Prefectural Agricultural School	Sendai ...	1905	4	6	485	Graduate of higher elem. schools.
	Iwate Prefectural Agricultural School	Morioka					do.
	Aomori Prefectural Agricultural School	Aomori ...	1848	3	6	488	do.
	Hokkaido Prefectural Agricultural School	Iwamizama	1907	3	6	293	do.
	Osaka Prefectural Agricultural School	Osaka ...	1888	4	6	566	do.
	Okayama Prefectural Agricultural School	Takamatsu	1899	4	4		do.
	Hiroshima Prefectural Agricultural School	Saijo ...					do.
	Yamaguchi Prefectrl. Agricultural School	Ogori ...					do.
	Oita Prefecture Agricultural School	Miye ...	1894	3	3	599	do.
	Kumamoto Prefectrl. Agricultural School	Miyagi ...	1900	3	4	225	do.
	Miyasaki Prefectural Agricultural School	Takanabe					do.
	Kagoshima Prefectrl. Agricultural School	Kanoya	1800	3	6	400	do.
	Nagano Prefectural Agricultural School	Iwamurata					do.
	Hokkaido Prefectural Agricultural School	Obihiro ...					do.
Private.	Tokyo Azabu Veterinary School ...	Tokyo ...	1890	3	18	2350	do.
	Tokyo Veterinary School	Tokyo ...	1907	3	19	1539	do.
	Nihon Veterinary School	Tokyo ...	1911	3	16	914	do.

The examination is given once a year.

The veterinary schools in the country are shown in the table.

The courses of the foregoing schools comprise, besides the veterinary courses mentioned before, agriculture and necessary subjects from the zootechny courses for veterinary surgeons. A foreign language, usually English, is given in the Morioka Agricultural School and other veterinary schools of secondary grade.

The Army has a veterinary school for the supplementary education of army veterinary officers, farrier non-commissioned officers and farriers. The veterinary officers are appointed from the graduates of a university or veterinary schools of higher school grades.

CLINICAL NOTES.

Tuberculosis of the Horse.

By WILLIAM TWEED, M.R.C.V.S., D.V.S.M., Corporation Veterinary Department, Sheffield.

This clinical case is reported because of the infrequent occurrence of the above condition in equine practice.

The subject was a heavy Shire gelding eight years old. The history of the case is that he had been kept working at a railway station, truck hauling, since purchase for a period of three years and the period from the commencement of symptoms until slaughter extends over six months. The first symptom shown was slight cough, which seemed to respond to electuary treatment. Intermittent bronchial catarrh appeared later and the horse was put off work, but he always became a fastidious feeder and was in better health when kept working; he was, therefore, allowed to continue until the symptoms became more serious. The nasal discharge gradually became more pus-like; there was no bad odour from the nostrils and only on one occasion after exercise was the discharge noticed to be blood-stained.

Variations of temperature began—105° F. was recorded—but it had a tendency to remain at between 102 and 103° F.

During this time the horse developed stiffness in the neck and latterly would groan if his head was pushed to turn him in the box.

Tuberculosis was diagnosed and the horse was slaughtered.

The *post-mortem* examination showed the cervical vertebræ to be affected and fused together. In the apices of the lungs were small areas of white caseous matter and the bronchial tubes contained a large amount of the same material. The spleen was enlarged and the substance throughout was dotted with fibrous nodules—the largest ones on the surface having white star-like centres. One mesenteric gland only was slightly enlarged and the carcase was in a well-nourished condition, despite the lesions present.

Sections were made from the spleen nodules and tuberculosis was confirmed by microscopic examination.

Echinococcal Cysts in Camels.

Three interesting, if brief, references to the occurrence of echinococcal cysts in camels have appeared in recent numbers of the *British Medical Journal*:—

Dr. J. C. MILNE (Hurghada, Egypt), (*B.M.J.*, 1925, December 19th, p. 1206), reports the discovery of two cysts of *Taenia echinococcus* in the lungs of an old camel. The outer walls of the cysts were thick and fibrous, with a white, easily broken lining. One cyst was filled with a clear fluid; the other contained a gelatinous material, with some calcareous fragments, and numerous hooklets. Dr. Milne asks whether this condition is common in camels. He has found in medical textbooks no report of its occurrence in them.

Dr. G. W. SUDLOW (Stoke-on-Trent) writes (*B.M.J.*, 1926, January 9th, p. 76): "Whilst serving as a temporary officer in the R.A.M.C. I was stationed, in the summer of 1918, in the Kharga Oasis, Libyan Desert. One unit under my charge was a company of Bikanir Camel Corps. Their camels were Indian, not Egyptian. After several unaccountable deaths of camels in their lines the Special Service officer attached to them asked me one day to see a sick camel. It was a fine beast, as were they all, apparently in their prime, and much larger and finer than the Egyptian camel. The history was that it has been off its feed for a few days, and was obviously ill and in pain. It died that night, and next day I conducted a *post-mortem* examination—no mean task. I found practically every abdominal organ riddled with cysts, large and small. I sent specimens of liver and kidneys for bacteriological report, which in due course confirmed my diagnosis (*post-mortem*) of hydatid disease. In all I made five or six *post-mortem* examinations on similar cases occurring in their camel lines within a few weeks. In my series of cases I saw both pleural and peritoneal cavities involved by the disease, also lungs, liver, spleen, and kidneys. The Indian officers of the camel corps kept dogs, which had the run of the camp, and the surrounding desert abounded in jackals, so that the source of infection was not far to seek. The camels' fodder was tibbin; this was stacked in the open in the camel lines, and evidently was being urinated upon by the infected dogs and jackals."

Dr. J. BURTON CLELAND (Professor of Pathology, University of Adelaide) writes (*B.M.J.*, 1926, May 22nd, p. 890): "In an article entitled 'Trypanosomiasis and other Diseases in Camels' (*Transactions* of the eighth session of the Australasian Medical Congress, 1908, vol. ii, p. 305, and also Bulletin No. 34, Department of Agriculture, Western Australia, 1909) I have recorded the occurrence of hydatid cysts in four camels which had recently been imported into the north-western part of Australia from Karachi in India. The following is an extract from this paper:—

Other Animal Parasites found in the Imported Camels.

"'Hydatids (*Taenia echinococcus*).—In *post-mortem* examinations on four of the camels hydatid cysts were found in each. In one there were about a dozen cysts, some as large as a goose's egg, in the lungs. In the other three there were up to half a dozen, ranging to the size of a hen's egg. Single cysts were found in the livers of two. Two of the cysts were degenerating and becoming calcified. In one a large number of daughter cysts, about the size of a grain of sago, were present. In some brood capsules were conspicuous. Scolices were numerous, and also the rounded concentric bodies like starch grains. Considering the number of cysts present, and the fact that two were degenerating, it hardly seems possible that the camels can have gained the infection in Western Australia during a stay of five months, especially as, in the only two bullocks I have seen slaughtered here, the liver and lungs were unaffected, and stockmen here are not apparently aware of the presence of hydatids in this part.'"—T. H.

REVIEWS.

Two Books on Nematodes.

Professor Stiles, the doyen of American helminthology, ends his foreword to the undermentioned work by a thought addressed to an unborn helminthologist, as follows :—

"My dear unborn colleague :

"When you consult this book, you will find in it some views with which you will not agree. This will be the inevitable result of an advance in knowledge after the publication of this volume. And as you differ with the taxonomic views expressed here, so will your successors modify your views as their knowledge increases. These changes of view from generation to generation are inevitable unless science stagnates.

"This work by Yorke and Maplestone was written in order to save you time and to make you more efficient in your professional life. It has cost them many hours and days of patient labour. It has not been prepared in order to gain a reputation. It is a labour of love on their part, in order to help you, see that you do for the generation which follows you what Yorke and Maplestone have done for you."

Coming as this does, from such an eminent authority, any comments from the writer would be of small value. In the preface the authors state their reasons for undertaking the work. The main one was with the object of simplifying the process of identifying nematode parasites of vertebrates. In this they have been entirely successful, for with a knowledge of nematode morphology and a little technique, the student ought to be able to place any nematode worm from any vertebrate in its genus. A parasite can be placed in its phylum, its class, and natural order. Then by keys, definitions and figures one passes from super-family, to family, to sub-family and to genus. Each genus is figured and there are 307 illustrations, many of which consist of four, five, or six diagrams. Having arrived at the generic position of a specimen, there follows a list of the known species, and numbered references from which one can choose a work if one requires specific determinations.

The taxonomic portion of the work is followed by a complete index of generic names and their synonyms, of some fifteen pages ; an index of specific names and synonyms, running to fifty pages, and a list of some 750 references right up to the date of publication. In view of some of the problems of synonymy and the need for recent references, this last portion of the work is of no less value than the illustrated part.

The collection of all the nematodes parasitic upon all classes of vertebrates into one work gives a pleasing and broad view of the subject to those used to a few genera in half a dozen hosts.

Whilst the veterinary surgeon may regret the absence of references to individual species and their bionomics, the authors point out what an enormous task such a work would be. The book may be considered essential for veterinary libraries, research institutes and those who have to teach "worm" subjects in our schools.

It is to be hoped that a system is in existence whereby additions and alterations can be recorded, so that future editions will be as up-to-date and helpful.

[The Nematode Parasites of Vertebrates, by Warrington Yorke, M.D., and P. A. Maplestone, M.D., D.S.O., with a foreword by C. W. Stiles. (J. and A. Churchill, London, 1926. Price 36s.)].

* * * *

The second work, unlike the preceding, deals with all nematodes, parasitic—both upon vertebrates and invertebrates, and free living forms. It is unillustrated but gives the authors' outline of a complete classification of the nematoda with the definitions of the natural orders, families, sub-families genera, etc. What Yorke and Maplestone regard as super-families are considered by the authors to be orders, save that in the present work the whole of the class falls into five orders, instead of eight super-families, for only the nematodes parasitic in vertebrates. The book will appeal to the systematic worker as there is nothing as complete between two covers. It will be of great help to marine, soil and plant workers.

[A Synopsis of the Families and Genera of Nematoda, by H. A. Bayliss, M.A., D.Sc., and R. Daubney, M.Sc., M.R.C.V.S., London. (Printed by Order of the Trustees of the British Museum, 1926. Price 10s. 6d.)].

A. W. N. P.

REPORT.

The Lister Institute of Preventive Medicine.

"The report of the governing body of the Lister Institute of Preventive Medicine states that the total expenditure for 1925 was over £36,500, a decrease of nearly £1,000 on that for 1924. The sale of vaccines, serums, etc., reckoning the value of the stock in hand, had yielded over £31,000. The total income of the institute was nearly £51,000, and the balance, being excess over expenditure, was over £14,000. The main part of the report is concerned with the research work done in the various departments of the institute. Professor Ledingham, F.R.S., the head of the department of bacteriology, has continued his researches in variola, vaccinia, and avian molluscum during the year. Certain anomalous reactions to small-pox virus have been observed in rabbits, and it is believed that their further investigation may throw valuable light on the irregularities met with in converting small-pox to the vaccinal form—a transformation which involves increased virulence of the original virus for the rabbit and a diminished virulence for man. Dr. Arkwright has continued his observations on the variation of bacteria *in vitro*, with regard especially to *B. paratyphosus* A. He has obtained a number of variants, and his observations, when complete, may prove to have an important bearing on pathogenesis in acute infections in animals and man, and also in chronic disease and the carrier state. Dr. Schutze has been investigating the relative potency of three

recognised types of plague vaccine: (1) Haffkine's prophylactic, (2) an agar-grown heat-killed vaccine, and (3) the serum-agar prepared at the Lister Institute. It was found that of guinea-pigs immunised with the first two, 14 and 22 per cent. respectively were protected against a lethal dose of *B. pestis* subsequently administered, whereas 44 per cent. of those immunised with the Lister Institute vaccine were protected. In rats, however, all the vaccines afforded protection to some 90 per cent. The experiments conducted by Dr. R. G. White to obtain fresh evidence as to the effect of pasteurisation of milk on tubercle bacilli showed that naturally infected milk from cows with tuberculous udders or milk to which cultural tubercle bacilli or emulsions of tuberculous glands were added was rendered incapable of producing tuberculous infection in guinea-pigs after it had been exposed to a temperature of 62·5° C. for thirty minutes. Among the investigations pursued in the department of biochemistry were those of Professor Harden, director of the department, in association with other workers in it, on vitamins. One of the subjects that are being investigated by Professor Harden is the nature of the substance contained in yeast and many vegetable extracts, which has a powerful stimulating effect on growth, and is considered by many to belong to the class of vitamins. Using lemon juice as the source of this material it was found possible to concentrate it very considerably and to separate it from the antiscorbutic vitamin. Fat-soluble vitamins have been studied in the department of experimental pathology. The provision of a steady supply of young rats with a constant endowment of vitamin reserves is an essential preliminary to satisfactory work, but has proved difficult to attain. Miss Henderson Smith, who supervises the breeding of rats for nutritional work, found that although the diet was maintained constant throughout the year great variation in the animals' subsequent resistance to deficiency diets occurred. The disturbing factor has been tracked to the seasonal variations in the vitamin content of the fresh milk, which was an important constituent of the diet of the breeding stock. Constancy has now been obtained by employing the same preparation of dried milk throughout the year; it is now included in the diet of mothers during the last stage of pregnancy, and in that of mother and young from the time of birth until needed for experiment. A great deal of other work on vitamins is being carried on in the institute, especially on the fat-soluble vitamins, by various workers, including Miss Harriette Chick, Miss Roscoe, and Miss Ethel M. Luce. The National Collection of Type Cultures has been maintained and extended, and over 4,000 cultures were distributed during the year."—*British Medical Journal.*

VARYING EFFECT OF POISONS ON ANIMALS.

"The smallest snail will withstand more strychnin than an adult man. Many of the stronger cardiac poisons have no action whatever on insects. The rabbit can take more morphin than can a man fifty times the animal's weight. Doses of lead, nicotin, etc., sufficient to poison man fatally do not injure the goat. Amygdalin does not affect dogs, but it kills rabbits. The hedge-hog takes with apparent enjoyment a dose of cantharides that will kill several persons under excruciating pains. Whereas, the frog is extraordinarily susceptible to the digitalis poisons, they have no effect upon the toad."—*Amer. Journal Pharm.*

DIVISIONAL REPORTS.

Lincolnshire and District Division.

"BRITISH RAGWORT POISONING."

A formidable outbreak of the poisoning of horses and cattle by rag-wort, which occurred within the area of the Division, provided material and occasion for an interesting paper and discussion on the subject at a meeting held at the Angel Hotel, Peterborough, on Thursday, June 10th, 1926.

The President, Mr. H. Caulton Reeks, sen., occupied the chair, and there were also present Messrs. W. A. Dickinson (Crowland), F. L. Gooch (Stamford), T. Knowles (Wisbech)—the essayist of the afternoon,—W. W. Lang (Brigg), G. Lockwood (Peterborough), J. Poles (Peterborough), E. Porter-Smith (Lincoln), T. Runciman (Ely), E. Wardrop (Wisbech), and H. C. Reeks, junr. (Hon. Secretary).

Apologies for absence were announced from Messrs. T. B. Bindloss (Long Sutton), J. Caldwell (Spilsby), W. W. Grasby (Daventry), H. P. Standley (Norwich), and Major C. W. Townsend (Long Stanton).

The minutes of the previous meeting of the Division, which had been published in the *Record*, were confirmed.

The PRESIDENT then said: As there is no routine business for our consideration this afternoon, the first duty I have to perform—and it is a very sad one—is to refer to the death of Sir Stewart Stockman. I am sure that it would be the wish of every member here that we should pass a vote of condolence with the widow and family. I do not think that any words are necessary from me to emphasise how great the loss of Sir Stewart Stockman will be to the veterinary profession. He was a very able man and, added to his knowledge, he possessed firmness and courage, both qualities of a very essential nature to one holding the post which he filled with such credit to himself and honour to the profession.

It was agreed that a letter of sympathy be sent, the vote of condolence being passed *sub silentio*, the members standing.

New Member.—Mr. R. G. Nurse, of March, and Mr. W. J. Ironside (Loughborough), were elected to membership of the Division.

Nomination.—Mr. A. Scotson, of Market Rasen, was nominated for membership.

At this stage Mr. GOOCH made reference to the Jubilee Congress of the Royal Sanitary Institute, to be held in London in the early part of July. The veterinary inspectors' section, of which Professor Wooldridge and he had been appointed secretaries, gave promise of being exceptionally interesting, and he felt sure that, while the section was always well worth the active participation and support of the profession, it would prove so in quite an exceptional degree on this occasion.

British Rag-wort (Senecio) Poisoning.

Mr. T. Knowles then presented the following paper on the subject of British Rag-wort Poisoning of cattle and horses :—

I have been asked to read a paper on British Rag-wort Poisoning of cattle and horses. Mr. Reeks has kindly consented to open the discussion, as he was concerned with me in a rather large outbreak which puzzled both of us for some time. The outbreak accounted for, on one small farm, the loss of about 15 head of cattle and 8 horses. It was some considerable time before we got to the cause of the trouble as, on a neighbouring farm, lead arsenate fruit spray had been used and was seen to blow over on to the pasture where the animals were grazing immediately before they showed symptoms of the disease. The symptoms shown were similar in the cattle to those of chronic lead poisoning, and in the horses, at the commencement of the outbreak, to acute arsenical poisoning. Lead was found in minute quantities in the liver and other viscera of the cattle, and small quantities of arsenic in the stomach of the horses. Arsenic was found in the soil. It was eventually realised that not enough lead arsenate could have gone over to account for so many deaths, the arsenic was found to be a normal constituent of the soil, and the lead discovered in the viscera in small quantities was probably carried in the drinking water. It was found that there was an enormous quantity of rag-wort growing in the pastures, which had been grazed all winter, so that when all the stock was turned out in the spring there was little but this for them to eat. The weed had just been mown and left lying about the pasture in a half-dry state. I think that animals will not eat it in the green or fresh state. Hay that the animals had been fed on all winter was also badly contaminated with the weed. After many consultations and much reading up of toxicology, we came to the conclusion that this weed was the actual cause of the deaths.

Symptoms may take from one week to nine months to show themselves after the ingestion of the weed, and according to the quantities eaten. Cattle seem only to suffer from the chronic form, which lasts about four weeks before proving fatal.

The *symptoms* are :—Gradual loss of appetite, accompanied by wasting, cessation of rumination and lactation ; the passage of diminishing quantities of fæces, which are liquid and sanious ; violent straining, which seems to become reflex in character ; the rumen becomes impacted with a solid, concrete-like mass ; grating of teeth ; salivation ; grunting ; progressive paralysis ; in-coordination of movement ; blindness, the eye assuming a peacock blue colour, followed by deep coma of about two days' duration, and death. The temperature remains normal ; the pulse is hard and infrequent, respiration laboured and slow, and the mucous membranes are injected.

Post-mortem shows advanced emaciation, flesh white and bloodless, great enlargement and cirrhosis of the liver, impaction of the rumen, œdema and great thickening of the submucous coat of the abomasum (yellow in colour), inflammation of the mucous membrane of the small intestine and colon ; the formation of a yellow-coloured viscous peritoneal exudate, varying from one to four gallons in quantity; mesentery congested with blood ; eversion of the rectum ; congestion of the lungs, due to the animal assuming a recumbent position ; pericarditis with formation of about half a pint of yellow clear fluid ; œdema of the kidneys, and a congested, pulpy spleen.

In horses, there are two forms of the disease ; acute and chronic. In the acute form, symptoms are—great pain, delirium and excitement ; profuse perspiration ; loss of co-ordination of movement ; blindness ; occasionally, radial paralysis of one fore-leg ; purgation ; death rapidly supervening. Pulse weak and frequent ; respirations rapid ; temperature elevated to about 104° F. ; mucous membranes deeply injected. In the chronic form, the horse may live two months. There are gradual wasting and loss of appetite, which may become depraved ; inability to focus objects with the eye but no actual blindness, the animal walking into things ; passage of diminishing quantities of fæces, which remain firm ; laboured, slow breathing and great use made of the abdominal muscles ; dilatation of the nostrils, with collection of food in posterior nares decomposing and discharging down the nostrils, the animal making a snoring noise ; yawning and very awkward when one wishes to examine the head and mouth. Temperature remains normal ; pulse hard and infrequent ; mucous membranes deeply injected. The animal gets down ; deep coma lasting about 36 hours supervenes and is followed by death.

Post-mortem appearances in the acute form—impaction and dilatation of the stomach (which is often ruptured), inflammation of the mucous lining of the small intestine and colon. In the chronic form—advanced emaciation, flesh white and bloodless, dilatation and impaction of the stomach, which is also ruptured in about 50 per cent. of cases ; enlargement and advanced cirrhosis of the liver ; thickening and œdema of the mucous membrane of the duodenum; remaining intestinal mucous membranes slightly inflamed ; pitechial marks on the peritoneum ; spleen pulpy and congested ; kidneys œdematous ; lungs congested but not gangrenous ; pericarditis as in cattle ; varying quantity (average about two quarts) of a treacle-like peritoneal exudate.

Treatment.—All kinds of treatment were tried and included large and repeated small doses of salines and oil ; stimulants both hypodermically and per os ; sedatives in the acute form, saturation with pot. iod. in chronic forms ; change of food and pasutre.

All these measures were, however, of no avail and the mortality was 100 per cent. Apparently, once rag-wort is eaten in sufficient quantities, nothing will expel the alkaloid, which may lie dormant for nearly a year before symptoms are actually shown.

I have had no cattle affected except on this one farm, but have had several horses on different farms since showing the chronic form, and in every case the pastures were badly contaminated with the weed.

This concludes my paper, gentlemen, and I thank you for your patient attention; my hope is that it has proved interesting and will provoke a good discussion, in which we may be able to learn something fresh about plant poisoning which, I am convinced, is much more prevalent than we suspect.

DISCUSSION.

The PRESIDENT: It appears—although I have no clear recollection of the point—that I gave some sort of undertaking to open the discussion. At the outset, therefore, I would say that I think we should thank Mr. Knowles very much for presenting to us a paper of such interest. As one who had considerable knowledge of the outbreak which he has described to us, I may say that the one point of criticism which I have to make is that he did not, perhaps, make it sufficiently clear in his paper what a very serious matter the outbreak was for those concerned. I happen to have before me a list of the animals that died, and when you come to reflect that the owner was only a smallholder, you will realise what a grave thing it was for him. On March 18th, 1923, the first horse died, the *post-mortem* showing a ruptured stomach. On May 2nd, a second horse died, when it was supposed that lightning was the cause. These cases occurred on the off farm. On May 6th or 7th, trees at the home farm were sprayed. On the 14th a cow died on the away farm (Sutton)—peritonitis being revealed on *post-mortem*. On May 17th, at the home farm (Parson Drove) a cow and a bullock died, no *post-mortem* being made. On June 7th at the same farm another bullock was lost, a *post-mortem* made and poison suspected. On this date the owner decided that if any more animals died he would have an analysis made. On the 12th of the month another bullock died. Following a *post-mortem* examination an analysis was made, which revealed the presence of lead. As a result all stock was moved to the away farm. On June 16th, however, a cow was lost at this farm, when the analysis report revealed the presence of lead. A week after the death of a bullock at Sutton, on July 3rd, the grass at both farms was analysed, arsenic being found in both. The stock was again moved to non-infected pasture and the infected pastures were definitely closed. The following details disclose subsequent events at the off and home farms.

Sutton—July 23rd, bullock died; *post-mortem* made.

Parson Drove—July 28th, nag horse at home place died. Analysis—arsenic.

Parson Drove—August 5th—bullock died; *post-mortem*.

Sutton—August 25th, cow died. Analysis—lead.

Sutton—December 5th, mare died; ruptured stomach. Analysis—lead.

Parson Drove—January 29th, 1925, bullock died. *Post-mortem* analysis—lead.

The total number of deaths was horses 4, cows and beasts 11.

On February 22nd, 1925, I was called in, at which time four horses were affected and seemed likely to die, and I think they have since died. At that time and for some time afterwards I was convinced that the cause of the trouble was the lead arsenate spray, but at the end I was just as convinced that it was nothing else but the rag-wort. I made sections of the liver and the cirrhosis was so marked that I do not think you could possibly mistake it. From a quantity of hay, about the size of a truss, I collected this amount of *Senecio* (produced) and all the mangers and racks were filled with the weed which the animals had rejected.

Mr. F. L. GOOCH: Where you get the chronic type of lead poisoning, such as is met with in cattle in this district, you find a distinct discoloration round the gums. It is quite a practice for people to buy up animals so affected for a sum in the neighbourhood of thirty shillings apiece. Four out of ten of them will die and the rest will make good cows.

Mr. E. WARDROP: Mention has been made of the symptoms as being like those of chronic lead poisoning, and I should like to ask if the essayist, or any other member present, has had experience of acute lead poisoning in cows. As many members will know, in a comparatively recent case in which I was interested, Professor Dixon, of Cambridge, stated that there was no such thing as acute lead poisoning. Great prominence was given to the case at the time and, mainly because of this statement by Professor Dixon, controversy on this question of the possibility or otherwise of the occurrence of acute lead poisoning in cattle was carried on in the professional press. In the course of the case, Major Hobday and Dr. Clough, Professor of Chemistry at Camden Town, gave evidence as to the existence of acute lead poisoning in cattle.

I made a *post-mortem* on a cow that died on August 25th and, at that time, I asked the lawyer if he would give me the privilege of sending parts of the cow to whatever analysts I liked to choose, receiving an affirmative reply. I failed to discover any symptom of lead poisoning, and on 26th August—the following day—I went down to the farm and examined the premises. I then came to the conclusion that these animals were poisoned through eating poisonous plants. A lot of this rag-wort was observable on a pasture that was almost devoid of grass. I told the lawyer for the defence that the best course for him to adopt would be to get a good botanist to examine the pasture. They went down a few days afterwards and I subsequently secured from the lawyer a copy of the report they sent in. As regards the arsenic and lead, one analyst found both to be present in the material forwarded from the cow which died on August 25th, and another found neither! That is a puzzling circumstance.

The PRESIDENT: It seems that there is more arsenic and lead in the soil here than I thought there was.

Mr. WARDROP: The most peculiar feature of the case was the difference in the two analyses. Since this case, quite recently, I have had others of the kind occurring. I have recently made a *post-mortem* on a beast which showed the same *post-mortem* appear-

ances as this cow, and when I examined the pasture I found any quantity of this rag-wort, but it is very hard to convince owners in our locality that this plant is the cause of the trouble.

The PRESIDENT: Undoubtedly; it has been there since their grandfathers' time and they will not admit that it is a serious source of danger to their stock.

Mr. WARDROP: These cases arise when the grass has been mown. The cattle have nothing else to eat and, although in the ordinary way they avoid it, they will then partake of it.

Mr. RUNCIMAN, sen.: I have certain cases cropping up nearly every year about which I am mystified. Two occurred last year in which the only symptom shown was that the animals could not swallow; in fact, they died of starvation. The trouble occurs at certain times of the year on certain pastures, and never a year passes without two or three deaths. Sometimes I have had a horse in which the history has been that he has suddenly started walking up and down the field—then make for a corner of the field and lie down. The owner knows it is going to die; it is evidently "off its head," but apart from this wandering about it has no maniacal symptoms. I had a case in which a horse was affected in his hind quarters, and when he backed he could not lift his legs, either front or hind. Animals so affected drag their limbs. The animal died, as the great majority of them do. It generally begins about this time of the year and on certain tracts of land. I have advised people to plough up these particular fields. The affected animals do not all betray the same symptoms—the affection is very much akin, in my opinion, to grass disease as it occurs in Scotland, but I have not observed the frothy sweating there. The only animal that did get better was a big shire mare, and it left her a "shiverer." What the complaint is, precisely, I am at a loss to know, but there is no doubt that it is plant poisoning of some kind. Cows are sometimes affected and the animals are not often seen until brain symptoms have developed, when they are dangerous to approach. They will knock the buildings about and die in twenty-four hours.

Mr. J. POLES: Are not symptoms of poisoning by ground ivy the same as those of poisoning by ragwort? Some years ago I saw some cases with Mr. Mackinder which exhibited similar symptoms to those described by Mr. Knowles, and he attributed them to the ingestion of ground ivy. The cattle affected were young—they went blind and showed considerable straining.

Mr. KNOWLES: Those cases seem to be of the acute type; these were more chronic.

Mr. POLES: I had some cases a fortnight ago. A man came to me one morning and said he had five young bullocks dead. I suspected black-leg, and I asked him where the bullocks were. The owner replied that he had had them taken away, but there was one at his place that was going to die. The eyes were injected and swollen; the bullock was down, but showed no pain whatever. This bullock died on the next day, and on *post-mortem* examination it looked like old-standing lung trouble. Both the lungs were only half the size that they should have been. The rumen was packed as full as it could be. The owner told me that the bullocks had been well all the winter, but a fortnight before he had turned them out to grass and they seemed to go back. He then put them into a paddock and, following that, into a very good pasture. They were only on the pasture two days when five were dead and he lost two more afterwards. The stomachs were packed with green stuff; all the organs were healthy except the lungs, and I cannot account for it.

At this stage the members discussed other interesting clinical topics, the main points of which will be summarised later. Reverting to the discussion on the paper—

Mr. G. LOCKWOOD said: I should like to ask the essayist if in his cases he found any symptoms of the animals going mad?

Receiving a negative reply, Mr. Lockwood proceeded: We had several cases of rag-wort poisoning in which, as soon as you got into the crew yard, the animals came for you, right across the yard. Another peculiarity is that on some farms you can find fields filled with rag-wort and yet you never get a case.

Mr. POLES: These are the cases in which the animals have nothing else to eat. I should like to ask which is the form in which rag-wort is the most dangerous, the green or the dry.

Mr. KNOWLES: The dry form.

Mr. GOOCH: I can tell you where you would get acute lead poisoning: by the side of the rifle ranges in the olden times when they used to have iron targets against which the bullets "splashed." We had several cases die at Stamford and some at Lincoln.

In the course of a discussion on other topics, Mr. LOCKWOOD said that he had had a mysterious experience amongst some young stock during the past winter, 21 dying between nine and fifteen months old. They were fed on 1½lbs. of cotton and linseed cake mixed, mangolds and hay. Five died for a start, and I thought the cause of the trouble to be the cake. We stopped that and all went well for a week, then eight more died. These animals showed practically no symptoms whatever, but *post-mortem* revealed acute inflammation of the fourth stomach and bowels. We had the water and everything else analysed, with the same experience as has been recounted this afternoon—one analysis showed lead in abundance in the water and the other showed none in it, both samples of water being obtained from exactly the same source.

The PRESIDENT: I believe that the condition to which you are referring is hæmorrhagic septicæmia. The ear is not a likely place in which to find it. The best way is to send blood from one of the heart cavities. I have met with four or five outbreaks of hæmorrhagic septicæmia during the last four years.

Mr. E. PORTER-SMITH: Have any of you met with hydrocyanic acid poisoning in young beasts? I had an alarming case in which the owner lost 17 beasts in 48 hours. It was proved that the poisoning was due

to the ingestion of cakes, and the owner received compensation.

Mr. GOOCH: I thought that it would interest the members to know that in Norfolk they frequently got animals bitten by adders, when the eye would swell up and the effect would pass off after 48 hours. Very few of the animals succumbed, the only ones to die being those bitten in the throat, when the swelling caused suffocation.

In reply to Mr. Lockwood, the PRESIDENT said that he had yet to see an animal recover from hæmorrhagic septicæmia.

Mr. POLES said that six weeks previously he had two interesting cases in cows. Both these animals went down with milk fever six days after calving. After apparent recovery they were turned out to grass, but within a fortnight precisely similar symptoms recurred, the usual treatment for milk fever was again adopted and they again recovered. Both the animals went down with the disease and recovered together. This happened three times; as soon as they were out to grass they seemed to "flush." He was wondering if these cases would throw any light on the problem of the causation of milk fever. He would like to know what was the best treatment in cases in which the cow was down, unable to rise, and showed symptoms of milk fever with the exception of the comatose condition. She was apparently healthy, but was unable to rise and some cases terminated fatally.

Mr. LANG: The best treatment is, I think, the administration of nux vomica.

Mr. POLES: I try nux vomica first, and if that is not sufficient I find it worth while to try liquor strychninæ.

Mr. DICKINSON: It seems to me that we get three classes of case. There is milk fever on the one side, post-parturient paralysis on the other, and in between you get cases in which the cow is partially unconscious, not feeding, not chewing the cud, and is moaning. They do not, however, relapse into total unconsciousness. These cases, in my experience, usually have a fatal termination. I invariably treat them for milk fever, inflating them two or three times. I have not used adrenalin in these cases.

Mr. LANG: I think it is quite possible that one can get a toxæmia, in such cases as those described by Mr. Dickinson, arising either from the stomach or the bowels. I had a case in which I got the animal up on the next day, but she went down again in the latter part of the second day. I blew her up again, but the third time I went I found she was in a worse condition than ever. I injected her with oxygen, gave a pound of salts and she recovered, never to look back. Toxæmia is one of the most common conditions with which we have to deal.

Mr. GOOCH: My father used to give a pint of turpentine in a quart of linseed oil for milk fever; you would not think he could do it without killing the cow, but I have seen him do scores and they recovered.

Mr. POLES: I have a little thing here, taken from a case which happened last week. It is a happening which you would not think possible. I was called to a cat which they said had something in its mouth. This is an ordinary hook, such as they put on meat from which to suspend a ticket, and it was right round the cat's tongue. It came through from the bottom, a good length was protruding from the middle upper surface of the tongue, and I cut it in half close to the tongue with a pair of scissors and pulled the pieces away. I cannot imagine how it could get into such a position.

Mr. LANG proposed a hearty vote of thanks to the essayist for his paper, and to the President for his conduct in the chair and for the able way in which he had introduced the discussion. Mr. Knowles' paper was one which he was sure would prove of very great interest to the profession.

Mr. GOOCH seconded the proposition, which was carried with acclamation.

H. C. REEKS, Hon. Secretary.

North Midland Division.*

"A FEW OF THE DUTIES OF A VETERINARY INSPECTOR."

A quarterly meeting of this Division was held at Sheffield on April 20th, 1926, when the following members were present:—Mr. C. Secker Smith (Barnsley), in the Chair, Messrs. Geo. Green (Rotherham), R. Hudson (Retford), E. F. Angler (Doncaster), F. C. Scott (Pontefract), W. Collinson (Unstone), E. Marrison (Bakewell), E. G. Johnson (Beighton), G. J. Furness (Alfreton), J. Abson, T. C. Fletcher, S. E. Sampson, W. Tweed, S. G. Abbott and A. McCrea (Sheffield).

Visitors.—Messrs. W. J. Ironside (Sutton Bonington), J. S. Woodrow (Goole), T. B. McClure (Londonderry), J. Holroyd, E. J. Burndred (Blackburn), J. McKinna (Huddersfield) and W. Woods (Wigan).

The meeting was to have been addressed by Professor Buxton, Cambridge, but owing to indisposition, Professor Buxton was not able to attend.

The minutes of the last quarterly meeting, having been printed and published, were taken as read and were signed as correct.

The minutes of the Council meetings held on February 12th and March 29th, respectively, were read by the Hon. Secretary, and on the motion of Mr. ABSON, seconded by Mr. COLLINSON, were adopted.

A letter was received from Mr. Robinson stating that as he was confined to his bed he was unable to be present. The President was asked to convey the sympathy of the members present to Mr. Robinson and also to send a letter of sympathy to Professor Buxton.

The HON. SECRETARY read a report of his attendance at the quarterly meeting of the National Veterinary Medical Association held in January, and Mr. MCKINNA gave a verbal report of what transpired at the quarterly meeting of the Association held in April.

* Received on June 26th.

Presidential Address.

The President then gave his address from the Chair.

At the outset the President thanked the members for the honour conferred upon him. He quite realised the great task set in following the gentlemen who had passed the Chair, but he would endeavour to carry out the duties of the office to their satisfaction.

The subject of his address would be "A Few of the Duties of a Veterinary Inspector," and, in the first place, he would say that the time was fast approaching when the appointment of wholetime veterinary inspectors in a great many towns would become an accomplished fact.

He knew, from his own experience, that the duties of the veterinary inspector were not easy or pleasant, and the responsibilities placed upon his shoulders were at times bound to cause him a great amount of worry and anxiety.

Mr. Smith went over briefly the chief diseases under the Contagious Diseases of Animals Act, *viz.*, anthrax, foot-and-mouth disease, glanders and farcy, parasitic mange, rabies, sheep scab, and swine fever.

In referring to tuberculosis, the President expressed the opinion that no measure had been passed that gave the veterinary inspector such a position and such power as the Tuberculosis Order, but he was sorry to say there were some members of the profession who abused it. It would be regrettable if they let the Government and the general public get the idea that they were too grasping.

Amongst many more duties of the veterinary inspector, Mr. Smith mentioned meat inspection, and maintained that this should be carried out by the veterinary inspector instead of by the sanitary inspector. He failed to see why the veterinary student, after studying meat inspection and obtaining his degree, should not be as fully qualified to examine meat as the man who obtained the certificate of the Royal Sanitary Institute.

In his younger days the duty causing him the greatest anxiety was the inspection of cattle markets, and this was due to the fact of his fearing that he might overlook something that might cause some serious results.

The inspection of cows and cowsheds should, in his opinion, be made quarterly instead of half-yearly; he gave this as a good example of the advantage which the whole-time inspector had over the part-time man.

The President claimed that the veterinary inspector was a real friend to the general practitioner because, if the latter was at all suspicious of any notifiable disease, he had only to report to the veterinary inspector and thus relieve himself of all anxiety and worry.

In concluding his address, Mr. Smith claimed that the veterinary inspector must be a man with the courage of his convictions and must carry out the duties without fear or favour. Any member of the profession appointed a veterinary inspector should be proud of his position, as it was a noble calling, because by far the greater part of the work done was for the benefit and welfare of his fellowmen.

The Hon. Secretary moved a vote of thanks to the President, which was seconded by Mr. Furness, who made some remarks on the sanitation of cowsheds, including air-space.

Mr. Hudson mentioned the difficulty of finding the acari of sheep-scab on sheep which had recently been dipped, and stated that in the diagnosis of anthrax at the place of death it was necessary to have a good light for the use of the microscope.

The vote of thanks was carried with acclamation.

As Professor Buxton was not present, the President invited a general discussion on matters appertaining to the profession and asked Mr. McKinna to open.

Mr. McKinna spoke upon the great necessity of cleanliness of cowsheds, cows and dairies in connection with the production of clean milk. He also stated the fees agreed upon by the Council of the N.V.M.A. for services under the Milk and Dairies Orders. He thought that the inspection should be carried out at least three times a year.

Other members gave their opinions as to fees.

Mr. Holroyd, President of the Lancashire Division, as a visitor, thanked the President for the invitation to be present, and said it had given him great pleasure to attend. He stated that in Lancashire inspection under the Tuberculosis Order was working smoothly in the hands of part-time inspectors and that their valuation was generally accepted by the owners. The greatest difficulty he had was in dealing with animals with chronic cough and showing definite signs of tuberculosis. These he valued at from £6 to £12, most of them being condemned and a quarter of the value paid as compensation. Owners did not like this procedure, as they could often sell such cattle to "slink" butchers for £3 or over, the butcher standing the risk. He thought the local authority should purchase the condemned animals at a valuation outright, as they stood. In dealing with tuberculous udders he was in favour of microscopic examination for tuberculosis bacilli in the milk and he thought the presence of alcohol acid-fast bacilli combined with the clinical symptoms of tuberculosis of the udder sufficient for condemnation. The tuberculin test was useful in cases of tuberculosis with emaciation, but in such cases the animals were not worth it.

Mr. Burndred, Blackburn, spoke on the inspection of contacts and said that this point in the Tuberculosis Order wanted definitely clearing up. He found, on *post-mortem*, that tuberculosis of the udder was generally present when the clinical symptoms of tuberculosis of the udder only were present. Advanced tuberculosis was the rule in regard to animals condemned under the Order. He drew attention to the requirements in regard to light and ventilation in cowsheds required by par. 5, section 11, of the draft Milk and Dairies Order, 1926, and stated that in his opinion veterinary surgeons should insist upon sufficient lighting and ventilation, together with cleanliness, so as to ensure as pure a milk supply

as possible. Veterinary surgeons in attendance should give advice in regard to such matters and try to get advanced farmers to lead their less advanced neighbours both by precept and example.

Mr. T. C. FLETCHER expressed the view that contamination of milk by retained placenta was a matter requiring serious consideration. What the owners usually did was to cut off the exposed portion and trust to the other being evacuated by the cow. He thought owners should be compelled to call a veterinary surgeon in such cases and that the placenta should be removed by hand.

(To be continued.)

NOTES AND NEWS.

The Editor will be glad to receive items of professional interest for inclusion in these columns.

July 27th-30th.—N.V.M.A. Congress at Dublin.

NEW MILK AND DAIRIES ORDER.

"A circular which is being issued by the Ministry of Health to county councils and sanitary authorities draws attention to the new Milk and Dairies Order and the Public Health (Imported Milk) Regulations, which supersede former dairies orders. It points out that the Milk and Dairies Order seeks 'to lay greater stress on cleanliness in all operations connected with the production and handling of milk (including the care of the cow) than upon the structure of buildings.'

"Local authorities are reminded that one veterinary officer may act under the Milk and Dairies Act and also under the Tuberculosis Order with convenience and economy. As to that part of the new Order which is enforceable by sanitary authorities, the Ministry remarks that officers appointed under the Sale of Food and Drugs Act 'will have opportunities for observing the conditions under which milk is consigned by rail (including the condition and marking of churns), and . . . where they have any reason to suspect any contravention of the provisions of the Order they should notify the responsible sanitary authority.' The need for co-operation between all authorities and their officers in the administration of the Order is emphasised."—*The Times.*

The Milk and Dairies Order, 1926, has now been made by the Minister of Health, with the concurrence of the Minister of Agriculture and Fisheries, and will come into operation generally on the 1st October, 1926.

The Public Health (Imported Milk) Regulations, 1926, have also been made by the Minister of Health, and will come into operation on the 1st January, 1927.

Both documents have been placed on sale, and may be purchased, price 3d. net and 1d. net respectively, directly or through any bookseller from His Majesty's Stationery Office at the following addresses:—Adastral House, Kingsway, London, W.C.2; 28 Abingdon Street, London, S.W.1; York Street, Manchester; 1 St. Andrew's Crescent, Cardiff.

TRAVELLING FACILITIES FOR THE DUBLIN CONGRESS.

Provided that a sufficiently large number of members advise us, arrangements can be made for a special saloon to be attached to the 8-30 a.m. boat train from London (Euston) to Holyhead on Monday, July 26th, for those attending the Dublin Congress. It is hoped that members intending to travel by this train will notify the General Secretary without delay, in order that the final arrangements may be carried through. Although the Association has not been able to secure special fares this year, Tourist Tickets, which are slightly cheaper than the Ordinary Tickets, can be obtained from most stations. Members can, however, verify this upon enquiry at their local stations.

HOLYHEAD AND KINGSTOWN MAIL SERVICE.

		Weekdays. Day Service. R.	Weekdays. Night Service. SS.	Sunday. Night Service. SS.	
		a.m.	p.m.	p.m.	
London (Euston)	... dep.	8.30	8.45	8.45	
		p.m.	a.m.	a.m.	
Chester ...	... arr.	12.15	12.35	12.35	
Chester ...	... dep.	12.23	12.43	12.43	
Holyhead ...	... arr.	2.7	2.25	2.25	
Holyhead Pier depart by steamer about ...		2.30	2.55	2.55	Mondays
Kingstown Pier	... arr.	5.25	5.50	5.50	Mondays
Dublin (Westland Row)	,,	6.0	6.35	6.35	Mondays

R=Restaurant Cars. SS=Sleeping saloons.

A LAY EDITOR ON "THE PROFESSION AND POOR PEOPLE'S CLINICS."

The Editor of *The Devon and Exeter Gazette* writes in the issue of his paper of July 5th:

"A copy of the current number of *The Veterinary Record* had been sent me containing a verbatim report of the discussion which took place at a recent meeting of the Central Division of the National Veterinary Medical Association on 'The Veterinary Profession and Poor People's Clinics.' The report is an extremely long one, and to give a resumé which would convey anything like a real impression would occupy too much space in this column or in any other section of the paper. I have, however, read the report fairly carefully during a short "rest" period, and believe I am fairly summing up the situation by saying that the general impression given during the discussion is that the profession does not condemn such institutions where the animals of those who are really financially unable to pay can receive treatment free. One of the members said, 'Nobody condemns them—one condemns, on behalf of the animals, the giving of medical and surgical advice and treatment by unskilled men when skilled men can be obtained.' I gather that the veterinary profession wants co-operation between themselves and such clinics, both in the interests of the animals and in order to prevent people who can afford treatment by a private qualified veterinary taking advantage of such institutions. If I have misconstrued the general feeling of the gathering I am open to correction, but the foregoing sentences seem to me to fairly represent a discussion which, from the verbatim report, was evidently carried on along broad-minded lines."

OBITUARY.

DAVIS.—On July 13th, at Lower Hutt, Wellington, New Zealand, Jeannie, wife of W. R. Davis, M.R.C.V.S. (late of Enfield, Middlesex), and eldest child of the late Principal Williams, New Veterinary College, Edinburgh.

SINCLAIR, George, Reezielaw, Newmachar, Aberdeen. Graduated Edinburgh, 29th May, 1890. Died *5th July*, 1926.

STAPLEY, Dr. Walter, M.D., Cambridge, New Zealand. Graduated London, 14th December, 1892. Died 22nd May, 1926.

CORRESPONDENCE.

Contagious Papillomatous Dermatitis of the Labia in Lambs.

TO THE EDITOR OF THE VETERINARY RECORD.

Sir,—The condition which Mr. Wallace Kerr has so admirably described and illustrated in your issue of the 3rd July, 1926, is of course the so-called *Aphtha*, or *Sore Mouth*. Descriptions, in detail, of the condition are very meagre in our text books although it is rather fully discussed in Rushworth's *Sheep Diseases*, but Mr. Kerr's is the most practical one. It is by no means uncommon in this district, and there are certain pastures where, if sheep (and I am referring to adult sheep in this connection) are turned out at certain seasons, an outbreak invariably occurs. A few years ago, I recorded an outbreak in *The Veterinary Journal* that occurred during a hot summer, in a flock that was on a pasture where there was a record "thistle crop"; and in this case the disease was attributed to the wounds made by the thistles, which were heavily infected with blight, the prickles directly inoculating it. I have since observed it in adult sheep and lambs out on roots, and on high and low-lying down pastures. Curious to relate, I have not observed it in the Cheviots. The mortality rate is usually low, although, according to Wallis Hoare, it may reach 10 per cent. in lambs. When the mouth lesions are severe it has a prejudicial effect upon the condition. Wallis Hoare (Vol. 2, p. 427) states that it has been attributed to *Oidium albicans*, whilst others attribute it to the *B. necrosis*. Rushworth (*Diseases of Sheep*, p. 162) states that the late Professor Nocard claimed that it was due to a special micro-organism which he found not only in the milk, but also in the watery effusions present in the abdominal cavity after death. I am inclined to believe that it is due to the spores of fungi that are present in certain pastures and that infection arises through the direct inoculation from small punctured wounds caused by the infected grasses or herbage. The condition is very contagious, and would appear to be sporadic. I have not tried Stockholm tar, but use a mouth wash, for mouth lesions, of *lin. saponis c. myrrh et benzoin* (*Hewlett*), and *liquor alumini acet.* (*diluted*) for lip and feet lesions, followed by *ung. acid boric c. lanoline*. The condition is not a hard one to cure, although some cases may be more troublesome than others. During the recent foot-and-mouth disease epidemic, I had more than one of these cases reported as "suspected foot-and-mouth."

I have heard that redweed will cause the condition, but of the correct botanical name of this I am ignorant.—Yours faithfully, J. F. D. TUTT.

1 St. Cross Road,
Winchester,
July 5th, 1926.

TO THE EDITOR OF THE VETERINARY RECORD.

Sir,—In your advertisement columns to-day there appears over my name a notice which, with your permission, I propose here to amplify a little. Let me give at once the origin of the idea of forming a Veterinary Practitioners' Association for Scotland. As many members of the profession know, there has been in existence for some time a group of part-time veterinary inspectors, to whom I have acted as secretary, who have interested themselves in the prospects of part-time inspectors under the Milk and Dairies (Scotland) Act of 1914. It has often been felt as a handicap by those who composed this group that they had no formal standing, and that when their secretary wrote to any outside body it was only by an act of grace that he could be considered as representing anybody but himself. To remedy this the idea naturally rose to form a permanent association, with officials regularly appointed, and whose activities would extend beyond the limits of the Milk and Dairies Act to include all suitable matters of interest to practitioners generally and not merely to part-time inspectors. That such a step should not be taken without the fullest consideration, the promoters have decided to call a meeting of practitioners, not necessarily to form such an association, but merely to consider whether or not it should be formed. The meeting is to be held in the Royal Hotel, 106 Saucniehall Street, Glasgow, on Saturday, 21st August, at 4 p.m., and it is hoped that as many practitioners as can do so will attend. Not only those who favour the idea, but also those who are opposed to it, so that the fullest discussion may take place and both sides of the case may be fairly stated.—I am, yours faithfully, DONALD CAMPBELL, M.R.C.V.S.

35 Calderwood Road,
Rutherglen.
13th July, 1926.

The Editor acknowledges receipt of the following:—
Communications from Mr. Fred Bullock, Capts. H. Burrell and T. M. Doyle.

National Veterinary Medical Association of Great Britain and Ireland.

EDITORIAL OFFICE: 10 Gray's Inn Square, London W.C.1.

All communications relative to the literary side of the Veterinary Record must be addressed to the Assistant Editor, "Veterinary Record," 10 Gray's Inn Square, W.C.1

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